

BEYOND BEHAVIOR: AN INTERSECTIONAL ANALYSIS OF THE IMPACT OF SEXUAL  
NETWORKS, SEGREGATION, AND INCARCERATION ON DISPARITIES IN STDS

BY

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DISSERTATION

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## ABSTRACT

There are approximately 19 million new cases of sexually transmitted diseases (STDs) in the United States every year (Satterwhite et al., 2008). Although the entire society is encumbered by the economic impact of STDs, the burden of these diseases is not equally borne along racial and gender lines. In particular, African Americans report substantially higher rates of chlamydia, gonorrhea, and syphilis than do Whites. Generally, women report higher rates of these diseases than do men.

Using an intersectional analytical framework, the first part of this research analyzes data from the 2006-2010 National Survey of Family Growth to examine the extent to which sexual network factors matter above and beyond individual-level sexual behavior and sociodemographic factors in accounting for STDs. In addition, it seeks to determine the degree to which sexual network factors account for the gaps in STDs among various race by gender groups. The results from multivariate logistic regression analyses suggest that, net of sociodemographic and behavioral factors, the main effects of the sexual network factors are consistent with the predictions derived from sexual network theory (i.e., four out of five hypotheses). However, factors associated with sexual network theory do not appear to go very far in terms of explaining the racial and gender gaps in STDs.

The second part of the research uses an intersectional analytical framework to assess factors consistent with an “American apartheid” perspective. It uses indicators compiled into a single dataset in which county (n=3,089) is the unit of analysis. The analysis examines the relationship of chlamydia rates and gonorrhea rates to racial isolation—a type of residential

segregation that measures the extent to which a member of a racial or ethnic group is likely to be in contact with members of this same group (as opposed to members of other groups). The analysis examines the relationship between both black isolation and white isolation and STD rates, net of other community-level factors that are associated with STDs. The analysis also compares the relationship of black isolation and white isolation to chlamydia rates for “white” counties, “integrated” counties, and “disproportionately black” counties. This research lends support to earlier studies that demonstrate that concentrated disadvantage through high black isolation is related to higher STD rates. But this research also adds to the literature by also showing that *white isolation* is associated with *lower* rates of chlamydia. Disparities in STD rates between disproportionately black counties and white counties would be greatly diminished if racial isolation were eliminated. Overall rates of chlamydia would decline by more than 33% if black isolation were eliminated. The results illustrate how powerfully residential segregation is related to STDs rates.

The third part of the research uses an intersectional analytical framework to examine the relationship between incarceration rates, being a community that serves as a reentry point for those formerly incarcerated, and STD rates. The analysis is based on county-level indicators compiled into a single dataset in which county (n=3,089) is the unit of analysis. The analysis examines the relationship of reentry locations and incarceration rates to STD rates, net of other community-level factors that are associated with STDs. These relationships are examined in disproportionately black communities, white communities, and integrated communities. The results suggest that communities that serve as reentry points for those who have been incarcerated have significantly higher rates of STDs, net of other community-level factors that

are associated with STDs. These reentry locations are more likely to be located in disproportionately black communities, and they help explain part of the relationship between racial isolation and STDs.

This study remains faithful to the tenets of intersectionality not only because it examines differences in race by gender groups, but also by taking into account residential segregation and contextual variables that are related to disparities in STDs. Moreover, this research looks at reentry locations as a way of measuring the impact of incarceration on the communities to which former prisoners return. This study is first to use national data to examine these factors in this way.

Persisting disparities in STDs in the U.S. is a stubborn problem that defies simple explanations. Differences in sexual behaviors do not account for the disparities. When sexual network factors are taken into account, the gaps become even larger. Community-level factors such as racial segregation and incarceration reentry location appear to hold promise.



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## TABLE OF CONTENTS

CHAPTER 1: RACE BY GENDER DISPARITIES IN CHLAMYDIA, GONORRHEA, AND SYPHILIS.....	1
CHAPTER 2: AN INTERSECTIONAL ANALYSIS OF SEXUAL NETWORK FACTORS IN THE REPORTING OF SEXUALLY TRANSMITTED DISEASES .....	21
CHAPTER 3: SEPARATE BUT UNEQUAL: RESIDENTIAL SEGREGATION AND SEXUALLY TRANSMITTED DISEASES .....	67
CHAPTER 4: INCARCERATION, EX-OFFENDER REENTRY, AND RACIAL DISPARITIES IN SEXUALLY TRANSMITTED DISEASES IN COMMUNITIES .....	108
CHAPTER 5: REVIEW OF FINDINGS, LIMITATIONS, CONCLUSIONS, AND IMPLICATIONS OF RESEARCH .....	138
REFERENCES .....	156
FIGURES AND CHARTS .....	183
TABLES .....	191
APPENDIX A .....	217
APPENDIX B .....	218
APPENDIX C .....	219
APPENDIX D .....	234
APPENDIX E .....	239
APPENDIX F .....	240
APPENDIX G .....	241

## **CHAPTER 1**

### **Race by Gender Disparities in Chlamydia, Gonorrhea, and Syphilis**

#### **The Problem**

In 2013, the Centers for Disease Control (CDC) reported that there are nearly 2 million known cases of chlamydia, gonorrhea, and syphilis combined. Chlamydia represented the most cases at 1.57 million, and syphilis represented the lowest number of cases with 117,000 (CDC, 2013A). According to the CDC, there is clear evidence that there are vast disparities in sexually transmitted diseases, both across racial and gender lines as well as between them. The CDC reports that African Americans have significantly higher rates of chlamydia, gonorrhea, and syphilis than do whites (CDC, 2013b). Although these STDs are often relatively asymptomatic and treatable, they can inflict dire consequences. This is particularly true for women, who may develop Pelvic Inflammatory Disease (PID)—“an infection of the uterus (womb), fallopian tubes (tubes that carry eggs from the ovaries to the uterus) and other reproductive organs that causes symptoms such as lower abdominal pain and infertility.” (CDC, 2013c).

According to LaVeist (2005), because of the vast increases in racial and ethnic minorities, the health of the U.S. will be a reflection of the health of these groups. Disparities in health are, therefore, reflective of the entire nation. The nation’s health collectively will be no stronger than the health outcomes present within minority groups (Rios and Rodriguez, 2008). It is, therefore, important to understand why such health disparities exist and how to reduce them.

The term “disparity” is used “to denote empirical differences between racial, gender, age, or sexuality groups” (Thomas et al., 2006: 25). When resources are not fairly distributed to those

groups that suffer from the disease, disparities can be viewed as unjust. When deciding which groups to compare, scholars have typically compared groups with more privilege to those that have been historically disadvantaged (Braverman, 2006:179). For the purposes of this study, health disparities in STDs will refer to the differences in STD rates between racial and gender groups with different social status in society.

Although the U.S. government has tracked the rates of disparities for more than a hundred years, health disparities did not become a central issue of study within mainstream sociology until the late 1980s (Williams and Sternthal, 2010: 51). One of the earliest sociological examinations of racial disparities in health is documented in W.E.B. DuBois' (1899) *The Philadelphia Negro* in which he suggests that "the higher level of poor health for blacks was one important indicator of racial inequality in the United States" (Williams and Sternthal, 2010: 50).

In order to understand the sources of health disparities, it is necessary to examine factors that are related to racial and gender disparities in other areas. Despite the vast amount of research and governmental gestures of support through outreach and education to reduce cases of STDs, these disparities persist. Despite the near eradication of syphilis through programs such as the Syphilis Eradication Effort (CDC, 2006) disparities in chlamydia, gonorrhea, and syphilis persist.

Why do African Americans face higher rates of STDs than do whites? This dissertation research seeks to answer several interrelated questions: (1) Above and beyond individual and behavioral determinants of STDs, what sexual network factors are specifically related to racial disparities in STDs? (2) Do sexual network factors account for disparities in STDs? (3) How is

residential segregation, especially racial isolation, related to racial disparities in STDs? And (4) how are incarceration rates and ex-offender reentry locations related to STDs?

### **Overview of Health Disparities in STDs**

According to Royer and Cerf (2009), academics and health care providers use the terms STDs and STIs interchangeably. However, some practitioners prefer to use the term STI with their patients because of the stigma associated with a disease versus an illness (Royer and Cerf, 2009:687). According to Eng and Butler (1997: 40), the “term ‘STD’ denotes the more than 25 infectious organisms that are transmitted through sexual activity, along with the dozens of clinical syndromes that they cause.” These diseases are disproportionately prevalent among those between the ages of 15 to 44 and racial and ethnic minorities (Eng and Butler, 1997). The Centers for Disease Control reports that there are “approximately 19 million new sexually transmitted diseases and infections (STDs and STIs) each year—almost half of them among young people ages 15 to 24” (Weinstock and Cates, 2007; CDC, 2012). Sexually transmitted diseases and infections carry a high economic and social cost. For example, the U.S. spends about \$15.6 billion annually on STD-related testing, treatment, outreach, education, lost time at work, and research activities (Owusu et al., 2013). There are also significant psychological and physiological cost such as stigma, fractured relationships, infant illness, and infertility (Chesson, Blandfor, Gift, Tao, and Irwin, 2004; CDC, 2009).

The reduction of sexually transmitted diseases has become a primary focus among health practitioners, academics, and the federal government. The federal government instituted the Healthy People 2010 initiative in order to eradicate all health disparities (Smedly et al., 2003).

This initiative suggests that “the health of the individual is almost inseparable from the health of the larger community, and ... the health of every community in every state and territory determines the overall health status of the nation” (Smedly et al., 2003: 37).

Despite the commitment of individuals, communities, and the government to the reduction of health disparities, such disparities persist, and the trajectory seems bleak with respect to sexually transmitted diseases. For example, African Americans suffer negative health outcomes at an excessive magnitude across most dimensions of health. Of the ten leading health disparities between African Americans and Whites, gonorrhea, syphilis, and new cases of AIDS top the list (Newman and Stuart, 2008: S4; Keppel, 2007).

STDs disparities are not new, and the scholarly literature notes extensively that STDs continue to plague African American communities at unjust rates (Laumann and Youm, 1999; Hogben and Leichter, 2008; Chesson, Kent, Kwame, Leichter, and Aral, 2012). Although all STDs, including HIV, are pressing issues, the next sections will discuss the differential rates of chlamydia, syphilis, and gonorrhea “because these STDs are the three most common, nationally reportable bacterial STDs” (Chesson et al., 2012: 1).

### **Chlamydia**

According to the Centers for Disease Control (2010: 7), chlamydia—“*C. trachomatis* infection is the most commonly reported notifiable disease in the United States. It is among the most prevalent of all STDs, and since 1994, has comprised the largest proportion of all STDs reported to CDC.” Sociologists suggest that health disparities, especially with respect to STDs are highly complex (Newman and Stuart, 2008: S6). The CDC (2006: 9) reports that although race and ethnicity do not fully account for STD disparities, they are tightly intertwined with other

social factors such as poverty, demographic factors, individual behavior, and structural issues. These factors make it difficult to pinpoint causal mechanisms. For example, in 2010, the disparity was such that African Americans were more than eight times more likely to have chlamydia than were Whites (1,167.5 and 138.7 cases per 100,000 people, respectively) (CDC, 2010: 8).

Similar disparities are also observed across gender and age. The CDC reported that chlamydia infection among women in all 50 states and the District of Columbia (610.6 cases per 100,000 females) was over two and a half times the rate among men (233.7 cases per 100,000 males) (CDC, 2010: 8). More startling is the conservative estimate that nearly 7 percent of all sexually active teenage girls have chlamydia (CDC, 2010).

According to the CDC (2012), chlamydia is particularly problematic because women are often asymptomatic. Given that many women fail to recognize symptoms, or have no symptoms at all, the disease often goes undiagnosed and untreated for many years. This leads to higher rates of transmission and complications related to the disease. The good news is that chlamydia is treatable with antibacterial medications; however, when left untreated it has a disproportionate impact on women and children. For example, women with untreated chlamydia are also at an increased risk for developing pelvic inflammatory disease (CDC, 2012). Pregnant women with chlamydia can also spread the infection to their babies during birth, leading to health complications (CDC, 2010). Similar to other STDs, some of the increase in chlamydia cases is due to better detection methods and higher reporting of positive cases to surveillance agencies (CDC, 2010). Despite these advancements in treatment and testing, African American women continue to face higher rates of infection.



## Syphilis

*Treponema pallidum*, also known as syphilis, progresses in three phases: (1) Primary syphilis, (2) Secondary syphilis, and (3) Tertiary syphilis (CDC, 2010). The majority of empirical studies examining rates of syphilis use surveillance data that have been reported based on positive results of cases in the first two stages of development (CDC, 2010). According to Newman and Stuart (2008: S6), “epidemiologists prefer to monitor syphilis that is in the earliest stages— that is, primary and secondary (P&S) syphilis; these cases would have been acquired recently and are better measures of disease incidence.” Positive cases of (P&S) syphilis are typically reported by STD clinics and, less often, by private practitioners (CDC, 2010). Although this reporting method has limitations, when estimating actual rates of syphilis in the United States, it continues to “provide a good indicator of the incidence of [the] disease” (Thomas, 1997: 47).

In 2010, (P&S) syphilis rates for African Americans were eight times the rate of Whites (28.2 cases per 100,000 vs. 4.0 cases per 100,000 (CDC, 2010).). Although this disparity is large, it is significantly less than the disparity “observed in 1999, when the rate among blacks was 24 times higher than the rate among whites” (CDC, 2010: 32). When reporting began in the 1940s, there were about 600,000 reported cases of late and latent syphilis (CDC, 2010:33). Syphilis rates have since decreased to less than 70,000 cases in the U.S. general population. This massive decrease in syphilis from the 1940s to 1999 encouraged lawmakers and health practitioners to develop the CDC’s National Plan to Eliminate syphilis (CDC, 2010: 33). Unfortunately, African American men and women have the highest rates of syphilis with 92.5 cases per 100,000 and

23.2 cases per 100,000 respectively. This compares with 1.8 cases per 100,000 for white women and 19.8 cases per 100,000 for white men (CDC, 2010: 2).

Syphilis, like other STDs, can have a significant deleterious impact on the health of an infected individual as well as an unborn child. “For example, up to 80 percent of pregnancies associated with untreated early syphilis result in stillbirth or clinical evidence of congenital syphilis in the newborn” (Eng and Butler, 1997: 47). Syphilis infection also increases the likelihood that an infected person would contract HIV if exposed. Over and Piot (1993) argue that by reducing 100 cases of syphilis, there would be the potential for a reduction of 1200 HIV infections linked to these cases within a ten year period.

The CDC (2006) suggests that “non-gay-identified” (NGI)-Men who have Sex with Men (MSM) who also have female partners likely contribute to P&S syphilis among women. Incarceration and geographic location may also play a significant part in the disparity between African Americans and Whites. According to Thomas (1997: 83), research has shown that among men in a Los Angeles County jail, rates of infectious syphilis were 11 times those of the general population.

There are many factors related to the racial disparity in syphilis. These factors involve biological, behavioral, and most importantly social structural forces (Thomas et al., 1999). Despite the overall decrease in the disparity within the general population, eliminating the disease remains an important goal for society.

### **Gonorrhea**

*Neisseria gonorrhoeae*, commonly known as gonorrhea, is “the second most commonly reported notifiable disease in the United States” (Barry, Kent, and Klausner, 2009: S62). Similar

to syphilis and chlamydia, empirical studies of gonorrhea tend to use surveillance data as their primary source. According to the CDC (2010), the STD Surveillance Network (SSuN) was established in 2005 in order to monitor changing trends in STD rates in the U.S. The SSuN collects information on STDs from 12 local and state health departments. The CDC also collects information on STDs from private reporting agencies, the National Health and Nutrition Examination Survey (NHANES), correctional facilities, emergency rooms, the National Job Training Program, and a host of other reporting agencies (CDC, 2010). The CDC compiles the data and releases it as annual STD surveillance reports. However, it is estimated that just about half of all positive gonorrhea cases are reported (Eng and Butler, 1997: 333). “Results from a U.S. physician survey showed low rates of routine screening and reporting of STDs from physicians, and presumptive treatment for gonorrhea without confirmatory tests was common” (Du et al., 2009: 444). Despite reporting limitations, surveillance data remains the best source of data to monitor changing rates of gonorrhea (Eng and Butler, 1997).

According to the CDC (2010: 17), there were 309,341 reported cases of gonorrhea. African Americans have the highest rates of gonorrhea, with rates that are reported to be 18.7 times (432.5 cases per 100,000 African Americans) the rate of Whites (23.1 per 100,000 Whites) (CDC, 2010: 17). In general, women have higher rates of gonorrhea than men. However, these rates vary by race. For example, in 2010 the gonorrhea rate for women was (106.5 cases per 100,000 females). The rate for men was (94.1 per 100,000 men). “The gonorrhea rate for Black men was 26 times higher than that in white men; the rate in black women was 17 times higher than that in white women” (CDC, 2010: 18).

Doctors and other health professionals began massive screening efforts in the 1970s (CDC, 2010). These screenings have had a significant impact on the health of women and their children. As with syphilis and chlamydia, gonorrhea is often asymptomatic and goes undetected for years. Untreated gonorrhea not only leads to an increase risk of infection of HIV and other STDs, but also increases the risk for developing PID, infertility, and ectopic pregnancy (CDC 2010: 17).

There is strong evidence that supports the notion that individual behaviors may lead to higher rates of gonorrhea. There is also compelling evidence that social structural factors are related to increased infections and may, in fact, matter more than individual behavior (CDC, 2010; Eng and Butler, 1997; Laumann and Youm, 1994). Researchers have found that incarceration, sexual networks to which individuals have access, SES of the community, access to health care, and race are all related to increased risk of gonorrhea infection (CDC, 2010; Chen, Ghani, and Edmunds, 2008: 441; Barry et al., 2009: S62; and Hogben and Leichter, 2008: S16). The next sections will focus on major theories used by sociologists and other researchers to help explain how these group-level and structural factors are related to chlamydia, syphilis, and gonorrhea.

### **Individual, Group Level, and Structural Explanations of STD Disparities**

According to Marable (1990), mainstream sociological research on African Americans is often framed by the cultural arguments presented in E. Franklin Frazier's work. Frazier (1957) characterized the African American family as moving toward a matriarchal structure leading to single female-headed households, countercultural values, and a general lack of self-control. This "culture of poverty" argument played a significant role in framing social policies concerning

education, economics, and health, especially after 1965. Overall, Marable (1990) notes that Frazier's claims concerning African Americans as primarily responsible for their own social condition (i.e., not being on par politically, socially, economically, or with respect to health outcomes with whites) helped to support racialized social policies that disadvantaged African Americans in the pre-civil rights era such as: Redlining, discrimination in G.I. benefits and segregation in education and in healthcare. These arguments were buttressed by the "Monyihan thesis" and shaped welfare and other social policies that continue to disenfranchise African Americans (Marable, 1990).

Despite challenges to cultural arguments, some contemporary scholars have continued to frame their analyses of racial health gaps around them (Hummer, 2006). Given the criticism of cultural arguments, why do scholars continue to argue that cultural factors matter with respect to the racial disparities in health? Cultural arguments were, in fact, responses to the question of why inequality existed despite legal changes in the status of African Americans after slavery and also to mounting evidence against biological deficiency theories. According to Alkalimat (1990), after 1965 the dominant ideological beliefs concerning racial inequality are that "Blacks' inferior status is largely attributable to blacks themselves, especially their lack of motivation .... Enough has been done." (p. 6). So how are these ideologies manifested within the literature on health disparities?

There are several factors related to disparities in STDs (Aral et al., 2005). These include but are not limited to behavioral factors, "incarceration rates, age composition, levels of education, income levels, segregation, unemployment, racism, sexual mixing patterns, and rates of concurrent sexual partners ('concurrency')" (CDC, 2006: 18).

The behavioral approach moves beyond the more racist biological arguments that justified the racial disparities in health (Hummer, 2006). However, behavioral research has often placed the responsibility of Black-White health disparities on the individuals. For example, Singh's (1990:56) review looking at infant mortality rates in the U.S. argues that:

the main barrier to prenatal care has to do with the behavior and motivation of mothers themselves .... A free society cannot force a woman to obtain prenatal care, nor can it constantly look over her shoulder to ensure that she doesn't smoke, take drugs, or otherwise damage herself and her child. Neither can the government, by itself, prevent out-of-wedlock births, stop young girls from having babies, or construct the supportive family setting necessary not only to the infant's survival of birth but also his survival of childhood and adolescence.

Such ideology places the onus on individuals, and it masks the larger social forces and group-level dynamics that place some individuals at greater risk despite their individual behaviors. It also hides the fact that there are structural forces that help shape group dynamics that place low-risk individuals at higher risk for a host of poor health outcomes including STDs. A personal responsibility perspective also hides other factors that relegate individuals to communities that expose them to higher risks of STDs compared with those in communities with lower risks (Thomas et al, 2010:102).

The general consensus among scholars and health professionals, however, is that individual-level behaviors fail to fully account for the racial and gender disparities in STDs. Scholars have put forth compelling arguments about the relationship of sexual network factors and racial disparities in STDs (Lauman and Youmm, 1999; and Adimora and Schoenbach, 2002, 2010). However, these studies have not examined whether the gaps in STDs between Black

women and other race by gender groups are reduced when accounting for sexual network factors. These studies also have not examined community-level factors and their role in maintaining racial disparities in STDs. Recently, scholars have looked at the relationship of residential segregation to STDs; however, they have not examined this by type of community. Given the call for empirical research looking at the impact of incarceration and reentry on communities, this dissertation will examine the relationship of STD disparities to sexual network factors, residential segregation, incarceration, and ex-offender reentry.

### **Intersectional Approach**

Researchers argue that when looking at health disparities there needs to be examination of complex relationship between individual, group, institutional, environmental, and political factors that help determine disparities in STDs (Hogben and Leichter, 2008). In this vein, the intersectional perspective demands the examination of individual and group level factors that help create and maintain disparities between black women and other race by gender groups, as well as, community level factors that are related to racial differences at the community level. Davis (2008), for example, highlights that intersectionality as a perspective assumes that different statuses intersect within institutions that shape individual outcomes.

Intersectionality as a perspective grew out of the absence of critical analysis of Black women's lived experiences. Intersectionality assumes simultaneity of race, class, and gender; yet, it does not ignore the fact that some statuses may take on more weight in given situations (Collins, 1998, and King, 1988). This study compares Black women to other social locations with respect to racial gaps in the likelihood of reporting of STDs. Although epidemiologists have looked at race by gender locations with respect to health disparities, this dissertation uses an

intersectional lens to focus on those disparities that plague African American women in particular. It also examines the relationship between community rates of STDs and structural forces such as residential segregation and incarceration, which place emphasis on the importance of contextual factors. In doing so, this research remains faithful to the tenants of intersectionality. This dissertation seeks to understand why African Americans, and Black women in particular, suffer the indignities of racial health disparities and how structural forces and phenomena beyond the individual preserve “two Americas, one healthy and white and the other filled with sick, disaffected people of color...” (Washington, 2006:12).

### **Medical Apartheid**

In her book, *Medical Apartheid: The Dark History of Medical Experimentation on the Black Americans From Colonial Times to the Present*, Harriet Washington (2006), argues that the implementation of the U.S. medical community’s treatments, research, and logics are not separate from the politics, economics, and ideologies of American society; rather, American medicine reflects it. The inequality in health outcomes between and among racial groups are, thus, reflective of the larger society’s inequality and the factors that sustain it (Washington, 2006).

The Tuskegee syphilis experiment serves as one of the most iconic STDs studies infused with racism, deception, and greed. The study spanned 40 years from the early 1930s to the 1970s. African American men were recruited and tracked during this time. The goal of the study was to learn how syphilis operates in people. However, the most dire of consequences were not fully observable until death. Therefore, medical scientists—with the approval and assistance of local, state, and federal agencies—prevented study participants from receiving treatment despite



having full knowledge that the disease was both treatable and fatal (Thomas and Quinn, 1991). Many of the arguments used to justify keeping the men in the dark about the goals of the study and from receiving treatment are still infused within the medical community today. For example, those running the study argued that:

black Alabamans [are] resistant to health measures, intellectually inferior, impetuous, degenerate, and above all, at the mercy of frighteningly powerful sexual drives...another fifty years will find an unsyphilitic negro a freak; unless some such procedure as vaccination comes to the relief of race and that in the hands of a compelling law (Washington, 2006:160).

Clearly, such racist logic has been proven inaccurate, especially with the success of the near elimination of syphilis among all groups through the use of penicillin rather than a vaccine. However, the underlying assumptions about black sexuality, promiscuity, blacks' unwillingness to participate in self-care and medical treatment continue to be woven into the fabric of explanations of racial disparities in general, and STDs in particular (Williams and Collins, 1995; and Collins, 1998).

Another major goal of this study is to examine factors related to racial disparities in STDs that shine the light on structural forces related to these disparities that are often ignored. LaVeist et al (2011) assert that research demonstrates the link between residential segregation and health inequality. Residential segregation for Blacks concentrates disadvantages and leads to worse outcomes in education, employment and health (Massy and Denton, 1993). Scholars suggest that place matters and that race often determines the lived environment where individuals work, go to school, have sex, and access healthcare. Therefore, examining the relationship between STD disparities and residential segregation is important.

Also, with the ongoing mass incarceration of Black men in the U.S., scholars need to examine the role of incarceration in all areas of social life. Many scholars argue that mass incarceration disrupts communities, familial patterns, and sexual relationships. These disruptions lead to sex ratio and power imbalances within the community. However, these disruptions do not end with incarceration. There is a cycle of reentry and re-incarceration that also places communities at risk for higher rates of STDs (Thomas and Thomas, 1999). With nearly 700,000 men leaving prison each year and about half of them returning within three years, the cycle of incarceration is clearly a problem for communities of color (Pettit and Lyons, 2009: 728). With prison as a new stopping point along the life trajectory for many low-income black youth (Pettit and Western, 2004), how incarceration and reentry affect the communities to which they are likely to return becomes an empirically important question.

This dissertation examines individual-behavioral factors, sexual network factors, racial segregation, and incarceration and ex-offender reentry as issues that are related to sexually transmitted diseases. It also examines the role of these factors in accounting for racial disparities in STDs. By examining these contextual factors, this research adds to a limited body of knowledge on the role of these community level variables and disparities in STDs. Below, I provide an overview of the remaining chapters of the dissertation.

### **Overview of the Chapters**

The central concern of this research is to go beyond individual behavioral factors that have been used to explain racial disparities in health outcomes such as sexually transmitted diseases. This study utilizes individual-level as well as community-level data to analyze whether factors suggested by the literature help account for the racial disparities in STDs. This research

includes individual-level characteristics such as age at first sex and drug use. In addition, it includes sociodemographic factors such as education, income, age, marital status, and immigrant status as part of a baseline model. It also includes other behavioral factors such as condom use and engagement in anal intercourse as control factors. These factors are in addition to variables related to sexual network models. Appendix G provides a chart that summarizes the hypotheses for the dissertation and indicates whether the analysis supported them.

Chapter 2 is entitled “An Intersectional Analysis of Sexual Network Factors in the Reporting of Sexually Transmitted Diseases.” It draws on an intersectional framework to examine whether there is a relationship between sexual network factors and the likelihood of individuals reporting an STD. The dataset used is the National Survey of Family Growth (NSFG). This is a national sample of 22,682 men and women “15-44 years of age living in households in the United States. Interviews were done 48 weeks of every year for 4 years—from June, 2006 through June, 2010” (CDC. 2012). This chapter seeks to determine whether sexual network factors help account for the likelihood of reporting STDs, whether they reduce the gap in reporting of STDs between Black women and other race by gender groups, and whether the groups vary in how sexual network factors are related to the likelihood of reporting STDs. Bivariate analysis is used to establish relationships between the central independent variables and the reporting of STDs. Because the dependent variable is dichotomous, a series of logistic regression models are used to test the hypotheses of interest. I hypothesize that the sexual network factors will be related to the increased likelihood of reporting STDs. The analysis also examines the degree to which sexual network factors help explain the gaps between Black women and other race by gender groups. It also carries out analysis to determine whether sexual

network factors vary in how they are related to the likelihood of reporting STDs for various race by gender groups (i.e., Black women compared with other race by gender groups). The chapter demonstrates that sexual network factors are an important part of the story of why people contract STDs. This chapter, like other studies, argues that individual behaviors and characteristics do not go far enough in explaining racial disparities in STDs. It illustrates the need for group-level factors and community-level factors that might serve to more fully unravel the puzzle of racial disparities in treatable STDs such as chlamydia, gonorrhea, and syphilis.

Chapter 3 is entitled “Separate but Unequal: Residential Segregation and Sexually Transmitted Diseases,” it introduces community-level factors and how they are related to racial disparities in STDs. Drawing on the literature dealing with racial residential segregation and hypersegregation, it examines the extent to which racial isolation is related to county-level rates of STDs. Given the call for the inclusion of contextual variables in the analysis of disparities in health outcomes (Zerai and Banks, 2002), particularly with respect to STDs (Chesson et al. 2004), I compiled a dataset that included indicators from all U.S. counties that reported data on chlamydia infections, gonorrhea infections, and racial composition (N=3089). It relies on indicators from various sources measured at the county level, including the Centers for Disease Control (2009 STD Surveillance System and the Bridged-Race Population Estimates), the U.S. Census Bureau (Counties Data Files, 2005-2007 and 2006-2010 American Community Surveys, and the Small Area Income and Poverty Estimates Program), and the Health Indicators Warehouse. These indicators have been compiled into a single dataset in which county is the unit of analysis. Indicators include the rate of gonorrhea infection per 100,000, the rate of chlamydia infection per 100,000 and residential segregation (i.e., the black isolation index and the white

isolation index). The purpose of this chapter is to examine the link between residential segregation (racial isolation) and STDs in various kinds of communities. This chapter looks at the relationship between both black isolation and white isolation and STDs rates in communities with different racial compositions. I use Ordinary Least Squares (OLS) regression models to assess the relationship between residential segregation and county-level STD rates. I hypothesize that in communities with the lowest percentages of Black residents, both black isolation and white isolation are associated with decreases in chlamydia rates and gonorrhea rates. In communities with intermediate percentages of Black residents, and in communities with the highest percentages of Black residents, net of other community factors, black isolation is associated with higher rates of chlamydia and gonorrhea, but white isolation is associated with lower rates of chlamydia and gonorrhea. This chapter posits that residential segregation is a factor that varies in its relationship to county-level rates of STDs and should be included in future research looking at STD rates.

Chapter 4 is entitled “Incarceration, Ex-Offender Reentry, and Racial Disparities in Sexually Transmitted Diseases in Communities.” It explores the relationship between incarceration, ex-offender locations (i.e., reentry), and racial disparities in STD rates in U.S. counties. It utilizes the same dataset as in Chapter 3. Using OLS regression models, I test whether higher incarceration rates in counties are related to higher rates of chlamydia and gonorrhea. I also test whether counties with ex-offender reentry facilities will have higher rates of chlamydia and gonorrhea compared with counties without reentry facilities. The research suggests the importance of residential segregation, incarceration, and reentry in their relationship to community rates of STDs.

Finally, Chapter 5 provides a recap of the study questions. It also discusses the hypotheses, and discussion of the findings. It also points out the limitations of the study, and it offers directions for future research. The United States is facing an often invisible crisis of racial disparities in STDs (Eng and Butler, 1997). Scholars have put forth compelling theories to help explain racial disparities in STDs; yet, the puzzle remains. This research extends the scholarly discussion of racial disparities in STDs. By using a nationally representative sample, this research examines sexual network factors related to racial disparities using an intersectional approach. This study remains faithful to the tenets of intersectionality theory by empirically testing the simultaneity of social location variables that may be related to disparities in STDs. The research also highlights the importance of moving beyond behavioral and sexual network factors to explain disparities in STDs between social location groups, and it demonstrates that sexual network factors overall do not operate qualitatively differently for Black women compared with other race by gender groups.

This dissertation also calls for further examination of the factors that maintain high rates of treatable STD gaps between African Americans and Whites. It demonstrates that group-level dynamics and structural factors are central to understanding the racial gaps in STDs. It argues that researchers must continue to push beyond the existing explanations and explore the extent to which these disparities affect different race by gender groups. It also sheds light on the relationship between residential segregation and STD rates in different types of communities. A central finding of the research uncovers how residential segregation is related to county-level rates of STDs. It also adds to the literature by examining the role of incarceration and ex-

offender reentry locations on county-level rates of STDs. It offers empirical evidence that makes a new case for the importance of the inclusion of these factors in future research.

## **CHAPTER 2**

### **An Intersectional Analysis of Sexual Network Factors in the Reporting of Sexually Transmitted Diseases**

#### **Introduction**

There are approximately 19 million new cases of sexually transmitted diseases (STDs) every year (Satterwhite et al., 2008). STDs impose physical, psychological, and economic costs on individuals and society as a whole. Women, who are frequently asymptomatic, face long-term consequences such as fertility problems and increased risks of other infections (Aral et al., 1996). The stigma associated with having an STD often discourages individuals from seeking treatment or informing partners of their status, which can lead to higher rates of infections within the population (Branson et al., 2006). Beyond the physical and emotional burdens caused by STDs, there is an extreme economic toll. In 2008, the estimated cost of STDs was \$15.6 billion, with chlamydia having the most economic impact of all bacterial infections (Owusu et al., 2013).

Although the entire society is encumbered by the economic impact of STDs, the burden of these diseases is not equally borne along racial or gender lines. For example, in 2010, Black women had the highest rates of chlamydia, and Black men had the highest rates of gonorrhea of all racial and gender groups (CDC, 2012). However, Latino men and Latina women also had higher rates of gonorrhea and chlamydia than whites (CDC, 2012). Despite these patterns, Gindi et al. (2010:191) indicate that “there were significant differences between Latinos and other racial/ethnic groups for several behavioral risk factors studied, with Latino patients reporting fewer behavioral risk factors than other patients.” Therefore, despite engaging in fewer behavioral risk factors, Latinos still have higher rates of STDs. Such disparities suggest that



something is wrong and cry out for a critical examination of the factors that help to create and maintain these inequalities. Therefore, this study will use an intersectional framework to examine those factors indicated by the behavioral and sexual network literature that are related to the likelihood of reporting an STD for different race by gender groups. It will include an analysis that compares the likelihood of reporting STDs among Black women, White women, Latino women, Other race women, Black men, White men, Latino men, and Other race men.

Davis (2008) provides a useful explanation of intersectionality and why it is useful.

Intersectionality refers to the interaction between gender, race, and other categories of difference in individual lives, social practices, institutional arrangements, and cultural ideologies and the outcomes of these interactions in terms of power. Originally coined by Kimberlé Crenshaw (1989), intersectionality was intended to address the fact that the experiences and struggles of women of colour fell between the cracks of both feminist and anti-racist discourse.

Crenshaw argued that theorists need to take both gender and race on board and show how they interact to shape the multiple dimensions of Black women's experiences (Davis, 2008:68).

Here, Davis (2008) highlights that intersectionality as a perspective assumes that different statuses intersect within institutions that shape individual outcomes. More importantly, intersectionality grew out of the absence of critical analysis of Black women's lived experiences. Intersectionality assumes simultaneity of race, class, and gender; yet, it does not ignore the fact that some statuses may take on more weight in given situations (Collins, 1998, and King, 1988).

Those concerned with studying racial and gender disparities in STDs should approach the subject with the understanding that individuals are not simply raced *or* gendered; rather,

individuals exist within a matrix of domination where they inhabit multiple identities that interlock in ways that affect individual outcomes in domains such as education, income, and health (Collins, 2009). According to Landry (2007:2), scholars studying inequality should be especially concerned with the intersection of race and gender because it has been among the “most important characteristics shaping lives and society in the US.”

Although intersectional theory is complex, it is a useful methodological tool for the examination of racial disparities in STDs. When analyzing these disparities through an intersectional framework, we make the assumption that not all women, or men for that matter, experience the sexual gender landscape in the same way. Landry (2007) asserts that individuals exist within simple social locations (i.e., race, class, or gender). However, because individuals also inhabit multiple locations that intersect, scholars must attempt to address this reality when analyzing differential outcomes. He suggests that when at least two simple locations intersect, they form “complex social locations” (Landry, 2007:217). This analysis will utilize the complex social location of race by gender. Throughout the dissertation, I refer to race by sex categories as “social locations” because this usage is consistent with the intersectional framework. In addition, intersectionality will be used as a method to examine the conditional (i.e., interactional) aspects of whether factors operate differently for various race by gender groups. For example, sexual network theory suggests that group level dynamics help maintain racial disparities in STDs (Laumann and Youm, 1999). However, an intersectional perspective suggests that we must also be mindful of the gender differences that occur within and between racial groups. Therefore, this analysis will use an intersectional approach to assess the extent to which sexual network theory accounts for STDs among different race by gender groups.

The disturbing trends in STDs disparities suggest that there are interactions between race and gender with respect to STDs. For example, although women generally have higher rates of STDs than men, researchers have not been able to unravel why African American men have higher rates of primary and secondary (P&S) Syphilis and Gonorrhea than do African American women. In addition, researchers have documented that biological differences between men and women contribute to gender disparities in STDs; however, biological differences fail to explain why there are racial disparities within gender groups (CDC, 2012). Clearly, such disparities highlight the fact that racial inequality is a continuing social problem. They also challenge colorblind notions of race that suggest that race is no longer relevant (Bell and Hartmann, 2007; Bonilla-Silva, 2010). However, Landry (2007) cautions against the taken-for-granted assumptions that intersecting fault lines of identity (e.g., race and gender) “actually” result in differential outcomes. He argues for empirical testing. These puzzles suggest that scholars must analyze the relationship between the interlocking systems of race and gender in order to more fully understand how these social forces help maintain health disparities in general and STD disparities in particular (Zeraï and Banks, 2002).

Intersectionality, as a theoretical perspective, is useful for this analysis because its tenets argue for a focus on Black women’s experiences. Intersectionality also critiques theoretical arguments that suggest Black women’s behavioral or cultural practices somehow result in their marginalization or disadvantage in society. Sexual network theories of STDs disparities posit that group-level dynamics such as concurrent partnerships, dissortative mating, and having high risk partners account for much of the disparity (Laumann and Youm, 1999). These theories often imply a cultural pathology argument, which suggests that if these individuals were in different,

less problematic networks, then their risk would be different. However, if such group level differences are key, then after accounting for these sexual network factors, disparities among race by gender groups should disappear.

### **Individual and Behavioral Factors and STDs**

Research indicates that behavioral factors matter in racial disparities in STDs; however, racial disparities persist even after accounting for differences in behavioral patterns (Hallfors et al., 2007; Singer et al., 2006; Pisani, 2008; Ellan et al., 1998; Aral et al., 1996; Newman and Berman, 2008). When attempting to explain racial disparities in STDs, we must control for factors that have been shown to be directly related to acquiring an STD. Although the data for this chapter does not allow for the examination of specific biological factors associated with gender (i.e., biological sex) disparities in STDs, I acknowledge that such factors do matter. For example, the physiological and anatomical differences between men and women place women at a disadvantage with respect to STDs. According to the CDC (2013), men are typically less vulnerable to STDs compared with women because women's vaginas provide environments that are dark and moist, that often have a thin lining that help support the growth and transmission of bacteria. In contrast, men's penises have one small opening and thicker skin. Particularly, men who are circumcised have reduced chances of contracting STDs compared with women (CDC, 2013).

Those concerned with population health and STDs have often focused their attention on individual-level behavioral factors that increase the risk of transmission (Aral et al., 1996). There are several individual-level characteristics that are related to an individual's likelihood of contracting an STD. Such factors include age at first sex, marital status, drug use, condom use,

anal sex, and engaging in homosexual sex (Aral et al., 1996). In her book, *The Wisdom of Whores: Bureaucrats, Brothels, and the Business of AIDS*, Pasani (2008) argues that HIV and other STDs disparities are directly related to behavioral factors such as condom use and using clean needles when taking illicit drugs. Pasani (2008) suggests that public policy interventions such as providing condoms, clean needles, testing, treatment, and the dissemination of accurate public health information would significantly reduce the disparities and overall rates of STDs.

Much of the literature suggests that individual behavioral factors that increase risks of STDs cluster together, particularly among adolescents (Auslander et al., 2009). For example, (Auslander et al., 2009:38) “found that adolescent girls engage in a range of sexual behaviors, with cultural differences in their choices of which behaviors to engage in and when .... [S]exual behaviors should not be considered in isolation, but rather as a pattern of behaviors that constitute a ‘sexual lifestyle.’” Salazar et al. (2009) found that among African American adolescent females with HIV, engaging in multiple sexual behavioral factors such as oral, anal, and vaginal sex is reported to be linked to an increased risk of STDs. They note that those who were “doing it all” were also more likely to report sex with someone who was formerly incarcerated and at least 5 years older. Beadnull et al. (2005) also suggest that it is important to analyze multiple behaviors to find a pattern of risky behavior because in some cases one type of behavior, such as sporadic condom use, is related to STDs and at other times it is not correlated. Bednull et al. (2005) argues that if these behavioral factors are addressed, the disparities would be greatly reduced. In this section, this chapter will provide a rationale for including these behavioral factors as controls (i.e., as baseline variables) within the analysis that examines sexual network factors.

### **Income and Education (SES)**

One of the most highly cited facts in the health disparities literature is the relationship between low socioeconomic status (SES) and negative health outcomes (House and Williams, 2000). Despite the extensive literature on the subject, there remain unanswered questions concerning how and in what direction the relationship between SES and health works (LaVeist, 2005; Williams and Collins, 1995). Williams and Collins (1995: 352) argue that health outcomes improve as SES increases and that those with lower levels of SES tend to have worse health outcomes.

With respect to STDs, scholars have assumed that there is a relationship between SES and STDs (Santelli et al., 2000). Research also suggests that the poor are more likely to be uninsured (Waldrop, 2000); thus, they are less likely to receive treatment, and women who are uninsured are less likely to receive reproductive or STD services than are insured women (Wyn, Ojeda, Ranji, & Salfanico, 2004). Datta et al. (2007), who used data from the National Health and Nutrition Examination Survey of individuals 14-39 years old, examined rates of chlamydia and gonorrhea in the U.S. and found that those reporting less than \$20,000 of household income were 1.8 times more likely to test positive for an STD compared with those making more than \$20,000 net of other factors.

Although there is a link between SES and STDs, much research suggests that there is a weak association between SES and racial disparities in STDs (Santelli et al., 2000; Newbern et al., 2004; and Biello et al., 2010). However, the research does suggest that there is a link between income disparities among households and racial disparities in STDs. There are also links between education and health outcomes. Because research suggests that there is a relationship

between SES and STDs in general, this analysis will include income and education as part of the baseline sexual network model.

### **Age and Age at Sexual Debut**

The literature suggests that age is a determinant of STDs (Kaestle et al., 2005; Nicollai et al. 2004). African American youth have been found to be at a higher risk of early sexual debut and at an increased risk for STDs (Upchurch et al. 1998). According to Dolcini et al. (1993), those who are younger are more likely to report having multiple sex partners, which is related to STDs. According to the CDC (2009), teens and young adults are far more likely to report chlamydia and gonorrhea compared with their sexually active adult counterparts. As noted earlier, women, and adolescent girls in particular, are also biologically more susceptible to STDs because of cervical ectopic—"the condition when columnar cells from the endocervix are present on the ectocervix, and thus more susceptible to infection. In particular, columnar cells are more likely to be infected by chlamydia, gonorrhea, and certain forms of HPV (most HPV strains preferentially infect squamous cells)" (Bosky, 2010; Aral, 2001). This places adolescent females at an increased risk of STDs compared with their male counterparts. Given the relationship between age and STDs, the analysis will control for age in the baseline as part of the sexual network analysis.

The age at which one becomes sexually active is also related to risk of contracting an STD (Beadnull et al., 2005; Kaestle et al., 2005). Using data from the National Youth Risk Behavior Survey (NYRBS) of youth ages 15 to 24 years, Eaton et al. (2009) found that there are racial and gender differences in reporting early sexual debut (i.e., before the age of 13). For example, African Americans report the highest rates of early sexual debut

with 24% of Black males and 5.6% of Black females reporting, this compares with 4.4% of White males, 2.2% of White females, and 9.8% of Latino males, and 3.7% Latino females Eaton et al. (2009).

Some scholars suggest that sexual debut is related to STDs among women because of biological susceptibility. For example:

Cervical ectopic (the presence on the exposed face of the cervix of a single layer of columnar cells that are typically found inside the cervix increases susceptibility to Chlamydia infection, HIV infection and perhaps to Gonorrhea. Cervical ectopic decreases with increasing age and smoking, and increases with hormonal contraceptive use. Thus, the risk for STD is increased among young women because of cervical ectopic, and delaying the age of sexual debut may be an important preventive behavior among women (Aral, 2001:212).

Although, the vaginal environment may increase the likelihood that a young woman would contract an STD, this biological susceptibility does not explain the racial disparity in STDs among women. Early sexual debut has also been cited as a leading risk factor for STDs among both young men and women, because it often occurs within a constellation of other risk factors such: drug use, inconsistent condom use, and multiple partners (Aral, 2001; Beadnull et al., 2005; Pflieger et al., 2013). Therefore, this analysis will control for age differences at first sex.

### **Marital Status**

Schoenborn (2004), using census data, found that more than half of U.S. residents are married. Marriage has generally been shown to be related to better health outcomes (Somers, 1979). According to Trease and Giesen (2000), fidelity is the primary practice among those who



are married. With roughly three percent of married individuals reporting infidelity (Trease and Giesen, 2000), marriage should provide protection from STDs.

According to Aral (2001:214), those who are unmarried living in the South are at an increased risk for STDs because they are more likely to have multiple sex partners. Using data from the National Survey of Family Growth 2002, Liddon et al. (2010) examined sexual risk among women of different marital statuses. They found that never married, divorced and separated, women were significantly more likely to report having more than five sexual partners in their lifetimes and two or more partners in the past year compared with married women. These findings suggest that marriage should be related to lower risk sexual behaviors; therefore, the analysis will include marital status as part of the baseline for the sexual network analysis.

### **Drug Use**

The literature often cites drug use as a behavioral risk factor associated with STDs (Aral, 1996). Drug and alcohol use has been associated with increased sexual risk-taking, particularly among adolescents, because it impairs judgment and places individuals at increased risk for STDs (Scott et al., 2009). According to Eaton et al. (2009), of those students reporting that they have had sex, 25% of males reported using drugs or alcohol before their last sexual encounter compared with 17.1% of females. Reports of drug and alcohol use before last sex also varied by race within gender groups, with whites reporting drug use before last sex more often than Blacks (Eaton et al., 2009).

Studies suggest that the relationship between drug use and STDs is also associated with reduction in condom use, multiple sexual partners, reduced condom negotiation for women, and increased reporting of STDs (Leigh, 2008; Pflieger et al., 2013). Despite the association between

drug and alcohol use, sexual risk-taking, and STDs, empirical evidence has not been conclusive in the matter with respect to causation (Scott et al., 2009; Leigh et al., 2008).

### **Condom Use**

Condom use has been found to be an important factor in reducing the transmission of STDs; however, inconsistent or inaccurate use does not provide adequate protection (Pflieger et al., 2013:e1). In their longitudinal study following students in grades 3 through 6 through grade 12, Beadnull et al. (2005) found that inconsistent condom use was related to reporting an STD. Eaton et al. (2009) also found that about 61% of those youth (10-24 years) in their study who were engaging in sex used condoms. Consistent with the literature, males reported higher condom use than females, with Black males reporting the highest rates (Eaton et al., 2009).

Pflieger et al. (2013:e1) note that, despite the protection provided by condom use, there are racial and gender differences in the consistent and accurate use of condoms, which can increase the risk of STD transmission among vulnerable groups. They also found that power differentials between men and women reduced the likelihood of women to report asking to use a condom compared with men, particularly among Hispanic women. However, Pflieger et al. (2013) also found that Black women reported higher condom use compared with white women; yet, they had higher STD rates. Pflieger et al. (2013) suggest that the higher observed STD rates among Black women may be related to sex ratios and higher infections rates within their sexual pools. The literature highlights the importance of controlling for condom use with respect to individual chances of contracting an STD; however, it also suggests that racial differences in condom use will not explain racial disparities in STDs.

### **Anal Sex**

Anal sex is widely accepted as a standard sexual practice and leading risk factor for STD transmission among men who have sex with men (Sanchez et al., 2006). However, reports of anal sex are increasing among those in the heterosexual population, and receptive anal sex is a known risk factor of STD transmission for this population (Leichliter et al., 2007). There are several reasons that anal sex is associated with higher transmission rates of STDs: (1) physical tears in and around the anus can expose both partners to bodily fluids that increase the chances of transmission; (2) heterosexuals engaging in anal sex may not view it as “sex” and, therefore, have a lower concern of pregnancy, which may lead to reduced condom use (Halperine, 1999); and (3) anal sex is associated with other high-risk behaviors that facilitate the transmission of STDs (i.e., drug use, reduced use of condoms, and sex with high-risk partners) (Samuel et al., 2010).

Jenness et al. (2010) used data from a sample of 436 women from high-risk areas in New York City to examine the relationship between unprotected anal sex and STDs. They found that those women who engaged in unprotected anal sex were 2.6 times more likely to report an STD than those women who engaged in unprotected vaginal intercourse (Samuel et al., 2010). Salazar et al. (2009) suggest that anal sex should be considered as a behavioral factor that increases the likelihood of contracting an STD. Other analysts suggest that engaging in anal sex may also be related to other high-risk sexual behaviors that can facilitate the transmission of STDs. For example, in their study of 202 young women ages 14 to 21, Auslander et al. (2009) wanted to analyze differences in oral and anal sex behaviors among girls of different races. They found that “those girls who had had both oral and vaginal sex were 6 times more likely to report having a sexually transmitted infection (STI) in the past compared to those who only had vaginal sex.

Those with oral, vaginal, and anal sex experience were 3 times more likely to report having an STI in the past compared to those who only had vaginal sex” (Auslander et al., 2009:35).

Auslander et al., (2009:37) also found that oral sex practices varied by race/ethnic but anal sex practices did not. Using data from Wave III of the National Longitudinal Study of Adolescent Health of young adult women Pflieger et al. (2013), categorized racial groups by low, moderate, and high risk groups. They found that sexual behaviors such as anal sex varied by racial groups. They showed that among the most common group of sexual risk behavior (i.e., moderate) Black women engaged in anal sex less often than did Whites; yet, they had higher rates of STDs.

Although black women are engaging in these behaviors less they still have higher rates of STDs, this can be related to other factors such as being in racially segregated pools and community-level factors such as residential segregation and incarceration. These studies show that engaging in anal sex for heterosexuals varies by race and gender, with whites reporting anal intercourse more often than Blacks, and men reporting anal sex more often than women (Leichter et al., 2007). These patterns also suggests that anal sex is related to STD transmission and should be statistically controlled when carrying out analysis even though it is not expected that anal sex patterns will account for disparities in reporting STDs among different race by gender groups. Therefore, this analysis will include participation in anal sex as a variable in the baseline model for the sexual network analysis.

### **Same Sex Relationships**

Engaging in homosexuality among men has been linked to an increased risk of HIV and other STDs (Millett, et al., 2006). According to the CDC (2009), there is an increasing trend of STDs among men who have sex with men (MSM) and men who have sex with both men and

women. The data suggest that in 2009, 62% of those with P&S syphilis were MSM (CDC, 2009). Studies have shown that behavioral risk factors associated with being MSM such as having multiple partners, having an increasing number of life time partners, and engaging in anal sex without a condom are also related to acquiring STDs (CDC, 2009). However, when controlling for these risk factors among MSM, racial disparities in STDs persist (Millet et al., 2006).

Despite the high rates of STDs among MSM, women who have sex with women (WSW) historically have been viewed as being at a decreased risk for STDs (Millet et al., 2006). Although the risks are low, there are documented cases of transmission of STDs among WSW (Marrazzo et al., 2005). In their study of 196 African American women who had sex with women and men (WSWM), Muzny et al. (2011) found that WSW in an STD clinic in Mississippi were less likely to have an STD compared with WSWM. They suggest that having sex with both men and women places the individual at an increased risk for infection. These studies suggest that homosexual behavioral should be taken into consideration when examining risk factors related STDs. However, it does not appear that homosexuality can account for racial disparities in STDs. Still, this analysis will include participation in same sex intercourse in the baseline model of the sexual network analysis.

### **Sexual Networks and STDs**

Behavioral factors related to STDs include the number of sexual partners an individual has, using condoms or not, type of sex (i.e., oral, anal, and vaginal), having sex with an intravenous drug users, selling or purchasing sex, and engaging in sex while under the influence of drugs and alcohol (Pisani, 2008). Although these factors are related to STDs, they fail to fully explain the racial disparity in STDs (CDC, 2006; Aral, 2005; Chesson et al., 2012; Hogben,

2008). Researchers and critical scholars must continue to ask if it is not just individual behavior, then “what is driving such disparities?” This failure of behavioral factors alone to explain STD disparities justifies a sociological examination of social and structural forces related to the racial and gender disparity in STDs.

Sociologists and epidemiologists have begun to highlight the role that sexual networks play in maintaining disparities in STD rates (Neman and Stuart, 2008; Salazar et al., 2009; Farley, 2006; Chesson et al., 2012). According to De et al. (2004:280) “a sexual network portrays the sexual inter-relationships within a defined group of people.” Some scholars argue that the sexual networks in which individuals have sex influence their likelihood of contracting an STD above and beyond their personal behaviors (Chesson et al., 2012; Farley, 2006). This section of the chapter provides further justification for examining sexual network factors that may be related to reporting an STD as well as those factors that may help account for racial disparities in STDs. An individual’s risk of contracting an STD is highly dependent on the rate at which the STD exists within the individual’s pool of sexual partners (Farley, 2006). The latter point is significant because an individual can engage in the most extreme and risky of sexual behaviors, but if the individuals with whom they have sex do not have an STD, they will not contract an STD. In contrast, if an individual practices safe sex in the form of a monogamous marriage relationship and their partner is infected, they are still at high risk for infection (Adimora et al., 2002). Thus, an individual’s risk of infection is related to the network in which they find themselves. Those interested in the sociology of health and medicine have turned their attention to social network theories to help explain racial disparities in STDs (Smith and Christakis, 2008).

Given that the determinants of individual health transcend personal behaviors, researchers have increasingly argued that the characteristics of one's sexual network partners and the networks in which individuals choose their partners significantly influence their chances of becoming infected (Laumann and Youm, 1999; Adimora et al., 2002). The literature suggests that sexual networks influence disparities in STDs because of intraracial mechanisms such as dissortative and assortative mating, as well as interracial mechanisms such as sexual isolation and sexual bridging (Laumann and Youm, 1999). These studies highlight the fact that sexual network factors matter above and beyond individual behavior (Smith and Christakis, 2008). The sexual network literature does focus on racial group disparities; however, few nationally representative studies have incorporated an intersectional analysis that examines whether these factors operate differently for men and women within and across racial groups (Mojola and Everett, 2012). Again, this study does so.

This analysis will examine both between and within racial group differences in the effects of sexual network factors on disparities in STDs. This chapter uses nationally representative data to examine whether factors put forth by sexual network theories matter above and beyond individual level factors when taking into account those unique social locations of race and gender. The specific sexual network factors examined are presented below followed by hypotheses suggesting the expected relationship between sexual network factors and reporting an STD.

## **Sexual Networks Factors as Determinants of Disparities in STDs**

### **Having Sex with Partners in Concurrent Relationships**

African Americans who engage in low-risk sexual behaviors such as having one sexual partner, often have partners who have concurrent sexual relationships (Laumann and Youm, 1999). For example, African Americans who report having one partner are 33 times more likely to have a partner who also has one partner, compared with those African Americans who report four or more partnerships. However, whites who report having one partner are 180 times more likely to have a partner who also has one partner, compared with whites who report four or more partners (Laumann and Youm, 1999:7). People engaging in what they think are low-risk relationships if their partners are in concurrent relationships and they are typically having sex more often and without condoms (Adimora and Schoenbach, 2010:1148). Adimora and Schoenbach (2010) argue that African American women because of sex ratio imbalances may be more tolerant of their partners being in concurrent relationships. Such imbalances may encourage acceptance of relationships with those in concurrent relationships. This may place African American women at risk for STDs because they feel less power to negotiate safe sex (i.e. condom use) because it infers infidelity (Adimora and Schoenbach, 2010). Such arguments places an emphasis on cultural behavioral practices with little attention on community level factors such as segregation and incarceration that may be related to higher rates of STDs within African American women's sexual pools. However, rather than condemning concurrency, which tends to be implicit in much of the sexual networks research, which appears more common among African American men, analysts should remain mindful that if these treatable diseases were reduced within the sex pools of participants, concurrent relationships, per se, would have



little effect on their risks of contracting STDs. Nevertheless, the sexual networks literature suggests that having sex with someone who has a concurrent sexual partner increases the risk of STD. So what role does a sexual partner's characteristic play in reporting STD?

*Hypothesis 1A:* Net of individual, behavioral, and other sexual network factors, those who have concurrent partners are more likely to report an STD.

*Hypothesis 1B:* Compared with African American women with concurrent partners, African American men are no more likely, but White women, White men, Latino women, Latino men, other race women and other race men with concurrent partners are less likely to report an STD, net of individual, behavioral, and other sexual network factors.

### **Having Sex with Risky Partners**

Studies that have utilized community and clinical data suggest that STDs are related to drug use (Flom et al., 2001). These studies argue that people who engage in drug and alcohol use are less likely to use condoms consistently, and thus, place themselves at risk for STDs, particularly among youth (Biello et al. 1994; Scott et al. 2009, Eaton et al. 2009). These studies also argue that particular drugs, like crack cocaine for example, increase the chances of engaging in high-risk sexual behavior such as having multiple partners, engaging in commercial sex, and reducing the use of condoms (Flom et al., 2001). For example, Flom et al. (2001) found that among those who reported using crack 64% of them had multiple partners, 84% of them had unprotected sex, and 60% of them reported having concurrent partners. Research also indicates that 9.5% of African Americans reported illicit drug use compared with 8.2% of Whites, and 6.6% of Latinos (Aldworth, 2009). These risky behaviors are sexual network characteristics because, even if people do not personally engage in these behaviors themselves, they can become

exposed to greater risks for STDs because others in their sexual network engage in these risky behaviors (Adimora and Schoenbach, 2005). Interestingly, research indicates that among those with STDs perceiving the actual sexual risk of their partner was not reliable (Stoner et al. 2003). The lack of an accurate assessment of one's sexual partner's risk adds another layer of complexity, to the role that sexual networks play in maintain disparities in STDs. Sexual network theory argues that those who are having sex with high-risk partners are at a greater risk for infection, and by extension, if African American women are more sexually isolated they are more likely to have sex with partners who engage in high risk behaviors than are whites, this should help account for the racial disparities in STDs.

*Hypothesis 2A:* Net of individual, behavioral, and other sexual network factors, those who engage in sex with high-risk partners are more likely to report an STD.

*Hypothesis 2B:* African American women with high risk partners are no more likely than are African American men , but more likely to report an STD compared with White women, White men, Latino women, Latino men, other race women and other race men with high risk partners, net of individual, behavioral, and other sexual network factors.

### **Core and Periphery Membership Statuses**

According to De et al. (2004:280) “a sexual network portrays the sexual inter-relationships within a defined group of people.” Some scholars argue that the sexual networks in which individuals have sex influence their likelihood of contracting an STD above and beyond their personal behaviors (Chesson et al., 2012; Farley, 2006). Laumann and Youm (1999), using data from the National Health and Social Life Survey (NHSLs), examined the effect of sexual networks on reported cases of STDs, net of behavioral factors such as being paid for sex, anal

sex, education, drug injection, and military service. They did not include “sex without a condom” as a variable because it did not remain relevant when conducting backward stepwise selection in multivariate modeling. Condom use has, however, been identified as a leading behavioral factor in the prevention of STDs (Pisani, 2008). Laumann and Youm (1999) found that gender was related to reporting an STD because women may have male partners who are more likely to have more female partners. With respect to racial disparities, they suggested that African Americans engage in highly “dissortative partnerships,” i.e., “mixing” relationships in which there is the partnering of low-risk members of a community with high-risk members. “Assortive partnerships” involve the mixing of individuals with similar STD risks (CDC, 2006: 10). Dissortative partnerships place low-risk individuals at high risk for infection. Those African Americans who are considered to be at the periphery because they have had only one partner in the last year were five times more likely to choose a partner who was at the core (i.e., had four or more partners in the past year) than were their White counterparts (CDC, 2006: 11). Dissortative mating has a significant impact on the racial disparities in STDs. Although, the data from the National Survey of Family Growth do not allow for the examination of whether individuals who are core members have sexual network partners who are periphery and vice versus, it does allow for the examination of whether an individual’s status as core or periphery. It is also possible that racial and gender differences in core and periphery membership statuses can influence the likelihood of reporting STDs differentially for race by gender groups.

*Hypothesis 3A:* Net of individual, behavioral, and other sexual network factors, core members are more likely to report an STD.

*Hypothesis 3B:* African American women core members are no more likely than are African American men, but are more likely to report an STD compared with White women, White men, Latino women, Latino men, other race women, and other race men who are core members net of individual, behavioral, and other sexual network factors.

*Hypothesis 4A:* Net of individual, behavioral, and other sexual network factors, periphery members are less likely to report an STD.

*Hypothesis 4B:* African American women periphery members are no more likely than are African American men, but are more likely to report an STD compared with White women, White men, Latino women, Latino men, other race women, and other race men who are periphery members, net of individual, behavioral, and other sexual network factors.

### **Racial Bridging**

“Bridging” exists when members of one group (e.g., African Americans, heterosexuals, etc.) have sex with members of another group, become infected, and then infect members of their own group (Hogben and Leichliter, 2008:S14). Laumann and Youm (1999) found that African Americans are less likely to engage in racial bridging because they are less likely to have sex with Whites than Whites are to have sex with individuals from other racial groups. Because African Americans’ sexual networks have higher rates of STDs, than do White sexual networks, this sexual network isolation of African Americans helps to maintain the disparities in STDs between Whites and African Americans by concentrating STDs within the African American population (Laumann and Youm, 1999).

*Hypothesis 5A:* Net of individual, behavioral, and other sexual network factors, individuals who engage in racial bridging are no more likely to report an STD compared with those who do not engage in racial bridging.

*Hypothesis 5B:* African American women who engage in racial bridging are no more likely than are African American men, but are less likely to report an STD, compared with White women, White men, Latino women, Latino men, other race women, and other race men who engage in racial bridging, net of individual, behavioral, and other sexual network factors.

Below, these hypotheses are tested with data from the National Survey of Family Growth. First, however, the chapter presents more information about the data and methods used in the analysis.

## **Data and Methods**

### **Sample**

The National Survey of Family Growth (NSFG) is a national sample of 10,403 men and 12,279 women (N = 22,682) between “15-44 years of age living in households in the United States. Interviews were done 48 weeks of every year for 4 years—from June, 2006 through June, 2010” (CDC, 2012). The “interviewing and data processing for the 2006–2010 NSFG were conducted by the University of Michigan’s Institute for Social Research, under a contract with the National Center for Health Statistics (NCHS). In-person interviews were conducted by trained professional interviewers in the homes of a national sample of households. Interviewers entered respondents’ answers directly into laptop computers. Interviews averaged about 80 minutes in length. The interview was voluntary; participants were provided information about the survey before being asked for signed informed consent. The survey was reviewed and

approved by the NCHS and University of Michigan Institutional Review Boards. The overall response rate was 75%” (CDC, 2012).

The national survey was based on an area probability sample that represents African Americans, Whites, Latinos, Asians, and others who are between 15 and 44 years of age. The “resulting sample was a nationally representative multistage area probability sample drawn from 85 areas across the country” (Mosher, 2010: 2-3). My study is explicitly focusing on the disparities between African Americans and Whites. However, it will also include Latinos and other racial groups in the models. “The sample is designed to produce national estimates and not state-specific estimates. Large areas (counties and cities) were selected first; then within each large area or PSU, groups of adjacent census blocks, called segments, were selected at random. In each segment, all addresses were listed, and some of the listed addresses were selected at random” (Chandra et al., 2012). NSFG has a complex non-random sample design; thus, I must include weights when conducting the analysis. The analysis used the `svyset` command within Stata 12.0 to appropriately weight the data: `svyset [pweight= wgtq1q16], strata (sest) psu(secu)`. This command makes the necessary adjustments for the fact that the 2006-2010 NSFG data are from a survey with a complex sampling framework. (CDC, 2012).

The data were collected through face-to-face interviews. However, more sensitive data were collected through Audio Computer Assisted Self- Interviewing (ACASI) technology. The program reads the question and the data contain information concerning sexual behaviors, demographic characteristics, and other risk factors that may be related to contracting STDs (CDC, 2012). Appendix A provides Pearson product-moment correlation coefficients for all pairs of variables used in the analyses (i.e., a correlation matrix).

There are other datasets that could be used to assess aspects of sexual network characteristics. For example, the National Longitudinal Study of Adolescent Health (AddHealth) has collected data on U.S. school age children in grades 7-12 in waves since 1995 (ICPSR, 2014). AddHealth is the most extensive survey that assesses network characteristics (Hatzenbuehler et al., 2012). It asks multiple questions about sexual partners' characteristics such as the age, race, level of romantic commitment, and types of sex intercourse (ICPSR, 2014). Therefore, these data are suitable for social network analysis that examines size and composition for connections, particularly when looking at concurrent partnerships (Morris et al., 2009: 1024). However, this survey does not ask questions about having sex with risky partners as does NSFG, which provides a comparable survey for testing hypotheses about concurrency (Morris et al., 2009: 1024). Moreover, because AddHealth only looks at those youth who are in grades 9-12, NSFG is a better data source because it also includes youth who are not in school, as well as people who are as old as age 44. Because these questions were not asked, it would not be possible to test the hypotheses concerning high-risk partners (Hypotheses 2A and 2B).

Another dataset known as the Youth Risk Behavioral Survey (YRBS) is a specialized survey developed by the CDC to complement the National Health Interview Survey. It has been collected every two years since 1991 by multiple national agencies and organizations along with the Center for Disease Control in order to track trends in behaviors among youth (9-12 graders) that lead to STDs, drug use, violence, and accidents (Brener et al., 2008, and Foti et al., 2011). The YRBS has also been used to examine sexual health behaviors. It collects information about demographics and sexual behaviors that are related to STDs. However, there are limitations with the YRBS. Although, the data is nationally representative it is limited to 9-12 graders. Thus, it

includes only those respondents who are currently enrolled in school. Moreover, it does not contain information on sexual partner characteristics necessary to test the hypotheses derived for this dissertation. Given the limitations of YRBS and AddHealth, NSFG is a more suitable dataset to utilize when testing the hypotheses concerning sexual network factors dealing with partner characteristics such as high-risk sexual partners and partners who have multiple partners.

### **Operationalizations**

STD. In order to measure STD, respondents were asked whether they had been told that they had gonorrhea, chlamydia, or syphilis. Respondents who responded yes to any of these diseases were coded 1 and others were coded 0.

Social location is the intersection of race by gender. To operationalize it, the race of the respondent was cross-classified by his or her gender and then dummy variable coded. To measure race of respondent, respondents were asked two questions: “Which of these groups ... best describes your racial background?” and “Now I have some questions about your ethnic background and your race ... Are you Hispanic or Latino, or of Spanish origin?” Respondents who reported being Hispanic were coded 1 (Hispanic) and 0 otherwise. Respondents who reported being black were dummy coded 1 if (black) and 0 otherwise. Respondents who reported being White were dummy coded 1 if (White) and 0 otherwise. Respondents of all other races were coded 1 (Other Race) and 0 otherwise. Female respondents were coded 1 and males were coded 0. The cross-classification yielded eight categories: (1) African American Females, (2) White Females, (3) Latina Females, (4) Other Race Females, (5) African American Males, (6) White Males, and (7) Latino Males, and (8) Other Race Males.



Education. To measure education, respondents were asked: “What is the highest grade or year of (regular) school you have ever attended?” Respondents were coded 9 for nine years (or fewer) through 19 (for 7 years or more of college).

Income. To measure income, respondents were asked: “Which category represents your total yearly income in the [prior] year, including income from all the sources ... such as wages, salaries, Social Security or retirement benefits, help from relatives, and so forth?” Responses were coded at the midpoint of the following categories: less than \$5000, \$5,000- \$7,4999, \$7,500-9,999, \$10,000-12,499, \$12,500-14,999, \$15,000-19,999, \$20,000-24,999, \$25,000-29,999, \$30,000-34,999, \$35,000-39,999, \$40,000-49,999, \$50,000- 59,999, \$60,000-74,999. Respondents who were reported \$75,000 or more were coded as \$90,000. This is consistent with the practice of adding a constant to the lower limit of the category that is equivalent to the range for the preceding category (Hout, 2004). Dollar amounts were recoded into ten-thousand dollar units by dividing by 10,000 to get more readable numbers.

Age. To measure the respondent’s age, respondents were asked: “How old are you?” They were coded for the actual age at the time of the survey.

Marital Status. Respondents were asked about their current marital statuses. Those who said they were currently married were coded 1, and others were coded 0. Those who were not currently married were also asked: ‘Do you live together with a [sexual] partner? By living together, I mean having a sexual relationship while sharing the same usual residence.’ Those who said that they are cohabiting were dummy variable coded 1, and others were coded 0.

Age at First Sex. To measure the respondent's age at first sex, respondents were asked when they first had sexual intercourse. They were coded for the actual age at the time of first sexual intercourse.

Same Sex. To measure same sex intercourse, respondents were asked: "Thinking about your entire life, how many same sex partners have you had?" Those who responded with anything other than never were coded 1 and others were coded 0.

Condom Use. In order to determine whether respondents used condoms during vaginal intercourse, they were asked: "Did you use a condom the last time you had vaginal/anal intercourse?" Those who said yes were coded 1, and all others were coded 0.

Drug Use. To gauge drug use, respondents were asked: "During the last 12 months, how often have you used cocaine/marijuana/crack/injection drugs?" Those who said never to all such questions were coded 0; those who said once or more during the year for any such illicit drug use were coded 1.

Anal Sex. To determine respondents' experiences with anal sex, they were asked: "Have you ever put/had a penis in a/your rectum or butt (also known as anal sex)?" Respondents who said yes were coded 1, and others were coded 0.

Concurrent Partners. To measure concurrent partners, respondents were asked: "In the last 12 months, did you have sex with any males/females who were also having sex with other people at around the same time?" Those who responded yes were coded 1, and others were coded 0.

Racial Bridge. In order to measure racial bridge, respondents were coded 1 if they reported having sex with a person (spouse/cohabiting partner or sexual partner) of a different race and 0 otherwise.

Risky Partner. Respondents were coded 1 if they reported having sex with a person with HIV, a person involved in prostitution, or a person who is an intravenous drug user. Other respondents were coded 0.

Core Members are respondents who have had four or more sexual partners in the past 12 months. Such respondents were coded 1, and others were coded 0.

Periphery Members are respondents who have had less than two sexual partners in the past 12 months. Such respondents were coded 1, and others were coded 0.

Social Location by Sexual Network Interactions are statistical combinations that describe the simultaneous influence of social location and sexual network factors on STD reporting such that the effects are multiplicative rather than additive. Appendix B provides a list of the social location by sexual network interactions.

## **Hypotheses**

As enumerated previously, the following hypotheses guide the analysis:

*Hypothesis 1A*: Net of individual, behavioral, and other sexual network factors, those who have concurrent partners are more likely to report an STD.

*Hypothesis 1B*: Compared with African American women with concurrent partners, African American men are no more likely, but White women, White men, Latino women, Latino men, other race women and other race men with concurrent partners are less likely to report an STD, net of individual, behavioral, and other sexual network factors.

*Hypothesis 2A:* Net of individual, behavioral, and other sexual network factors, those who engage in sex with high-risk partners are more likely to report an STD.

*Hypothesis 2B:* African American women with high risk partners are no more likely than are African American men , but more likely to report an STD compared with White women, White men, Latino women, Latino men, other race women and other race men with high risk partners, net of individual, behavioral, and other sexual network factors.

*Hypothesis 3A:* Net of individual, behavioral, and other sexual network factors, core members are more likely to report an STD.

*Hypothesis 3B:* African American women core members are no more likely than are African American men, but are more likely to report an STD compared with White women, White men, Latino women, Latino men, other race women, and other race men who are core members net of individual, behavioral, and other sexual network factors.

*Hypothesis 4A:* Net of individual, behavioral, and other sexual network factors, periphery members are likely to report an STD.

*Hypothesis 4B:* African American women periphery members are no more likely than are African American men, but are more likely to report an STD compared with White women, White men, Latino women, Latino men, other race women, and other race men who are periphery members, net of individual, behavioral, and other sexual network factors.

*Hypothesis 5A:* Net of individual, behavioral, and other sexual network factors, individuals who engage in racial bridging are no more likely to report an STD compared with those who do not engage in racial bridging.

*Hypothesis 5B:* African American women who engage in racial bridging are no more likely than are African American men, but are less likely to report an STD, compared with White women, White men, Latino women, Latino men, other race women, and other race men who engage in racial bridging, net of individual, behavioral, and other sexual network factors.

For each hypothesis, it shows how sexual network factors are related to STDs. Then it shows how these sexual network factors are related to social location disparities in STDs.

### **Analysis Strategy**

In order to test the hypotheses, the analysis consists of a series of logistic regression models in which the likelihood of reporting an STD (either chlamydia, syphilis, or gonorrhea) is the dependent variable. The intersectional framework calls for comparing the likelihoods of reporting such infections for White males, African American males, Latino males, other race males, White females, African American females, Latina females, and other race females. The baseline model includes such sociodemographic and behavioral factors such as income, education, age, sexuality, marital status, condom use, engagement in anal sexual intercourse, and illicit drug use. The analysis is ultimately concerned with examining the race by gender gaps in reporting STDs. In the multivariate analysis, African American women serve as the comparison group.

The following logistic regression model is estimated:

$$1. \ln[p(\text{report STD})/(1-p(\text{report STD}))] = \alpha + \beta_1 * \text{White Female} + \beta_2 * \text{Latina Female} + \beta_3 * \text{Other Race Female} + \beta_4 * \text{Black Male} + \beta_5 * \text{White Male} + \beta_6 * \text{Latino Male} + \beta_7 * \text{Other Race Male} + \beta_8 * \text{Education} + \beta_9 * \text{Income} + \beta_{10} * \text{Age} + \beta_{11} * \text{Married} + \beta_{12} * \text{Cohabiting} + \beta_{13} * \text{Age at Sexual Initiation} + \beta_{14} * \text{Used Condom} + \beta_{15} * \text{Uses Drugs} + \beta_{16} * \text{Anal Sex} + \beta_{17} * \text{Concurrent Partners} + \beta_{18} * \text{Risky Sexual Partner} + \beta_{19} * \text{Core} + \beta_{20} * \text{Periphery} + \beta_{21} * \text{Racial Bridge} + \varepsilon$$

This equation corresponds to Hypotheses 1A, 2A, 3A, 4A, and 5A. The model estimates the likelihood of reporting an STD when controlling for individual, behavioral, and sexual network factors.

$$2. \ln[p(\text{report STD})/(1-p(\text{report STD}))] = \alpha + \beta_1 * \text{White Female} + \beta_2 * \text{Latina Female} + \beta_3 * \text{Other Race Female} + \beta_4 * \text{Black Male} + \beta_5 * \text{White Male} + \beta_6 * \text{Latino Male} + \beta_7 * \text{Other Race Male} + \beta_8 * \text{Education} + \beta_9 * \text{Income} + \beta_{10} * \text{Age} + \beta_{11} * \text{Married} + \beta_{12} * \text{Cohabiting} + \beta_{13} * \text{Age at Sexual Initiation} + \beta_{14} * \text{Used Condom} + \beta_{15} * \text{Uses Drugs} + \beta_{16} * \text{Anal Sex} + \beta_{17} * \text{Concurrent Partners} + \beta_{18} * \text{Risky Sexual Partner} + \beta_{19} * \text{Core} + \beta_{20} * \text{Periphery} + \beta_{21} * \text{Racial Bridge} + \beta_{22} * (\text{White Female with Sexual Network Factor}) + \beta_{23} * (\text{Latina Female w}) + \beta_{24} * (\text{Other Race Female with Sexual Network Factor}) + \beta_{25} * (\text{Black Male with Sexual Network Factor}) + \beta_{26} * (\text{White Male with Sexual Network Factor}) + \beta_{27} * (\text{Latino Male with Sexual Network Factor}) + \beta_{28} * (\text{Other Race Male with Sexual Network Factor}) + \varepsilon$$

This equation corresponds to Hypotheses 1B, 2B, 3B, 4B, and 5B. The model estimates the likelihood of reporting an STD when controlling for individual, behavioral, and sexual network factors and the interactions between social locations and sexual network factors.

The social location by sexual network interaction model above provides information about whether the relationship between sexual network factors and reporting an STD varies by the category of social location. For example, if the coefficient for Latina women with concurrent partners is statistically significant, this suggests that the relationship between concurrency is different for Latina women and Black women. In order to facilitate assessment of Hypotheses 1B, 2B, 3B, 4B, and 5B, the results from the social location by sexual network interaction models will be transformed into predicted probabilities. This is accomplished by using the following formula from Liao (1994:12):

$$\text{Prob}(y = 1) = 1 - L \left( - \sum_{k=1}^K \beta_k x_k \right) = L \left( \sum_{k=1}^K \beta_k x_k \right) = \frac{e^{\sum_{k=1}^K \beta_k x_k}}{1 + e^{\sum_{k=1}^K \beta_k x_k}}.$$

This is accomplished by using Stata 12.0 and issuing the `prtab` command for each social location by sexual network combination.

The steps in this analysis strategy were repeated when carrying out analysis of reporting STDs among those who have been tested within the past 12 months (i.e., STD2). The results of that analysis are presented in Appendix C. However, only the results from the final social location by sexual network interaction models are reported.

### **Some Preliminary Diagnostics**

As part of the preliminary diagnostics, the analysis examined the goodness-of-fit of various models to determine whether the models of interest provided better fits than alternative models. Given that the NSFG data are complex survey data, Archer et al. (2006) propose using the Hosmer-Lemeshow goodness-of-fit test. This test was executed by using Stata 12.0 and issuing the `estat gof` command (Stata, 2013). According to Archer et al. (2006: 4463), “the hoped-for outcome from a goodness-of-fit test is to fail to reject the null hypothesis.” Therefore, any test that yields a  $\chi^2$  value greater than  $p=.05$  is assumed to be a good fit (Archer et al., 2006). For each of the models specified in the analysis, the  $\chi^2$  value for the Hosmer-Lemeshow goodness-of-fit test was greater than .05. It is acceptable to proceed with the models of interest.

In order to determine whether the independent variables were too highly correlated, I ran a correlation matrix (see Appendix A). The correlation matrix shows the Pearson product-moment correlation coefficients for all pairs of variables used in the analyses. It shows that there are 18,843 observations when using listwise deletion (i.e., the entire observation is omitted from the estimation sample if any of the variables in to be used in the analyses is missing for that observation) (Bruin, 2006). The correlation matrix shows that for all variables the correlations

are less than .5. Generally, this suggests that the independent variables to be used in the analyses are not too highly correlated so that they create problems of co-linearity (Kleinbaum et al., 1987). In addition, in the multivariate models, because the NSFG data are from a complex sample, I carried out the collin command in Stata 12.0 to assess the variance inflation factor (VIF). This test suggested that none of the independent variables were too highly correlated, as all values on the VIF were less than 10.0 (Institute for Digital Research and Education, 2012).

### **Results**

Table 2.1 presents a comparison of respondents with STDs to those without STDs. This table, created in Excel, presents the percentage distribution (i.e., composition) of those reporting an STD versus those not reporting an STD for each of the variables used in the multivariate analysis. It also presents a  $\chi^2$  statistic for each characteristic to show whether those with STDs differ systematically from those without STDs on the given characteristic. Generally, this table shows that those with STDs differ from those without STDs on several characteristics. In particular, they differ in terms of their racial and gender compositions. Black females comprise 23.6% of those with STDs but 6.9% of those without STDs. In contrast, White females make up 22.4% of those with STDs but 30.9% of those without STDs. Latina females are slightly underrepresented among those with STDs (5.8% versus 8.5% overall). Other race women are slightly overrepresented among those with STDs (4.3% versus 3.4% overall). Black males make up 16.2% of respondents with STDs but 6.1% of those without STDs. White males are 20.9% of those with STDs but 31.2% of those without STDs. Latino males are underrepresented among those with STDs (5.2% versus 9.6% overall). Other race men are underrepresented among those with STDs (1.7% versus 3.2% overall).



Table 2.1 also shows that those with STDs and those without STDs differ in terms of sexual network characteristics. Those with concurrent sexual partners comprise 9.1% of those without STDs but 34.8% of those with STDs. Those with risky sexual partners make up 1.8% of those without STDs but 13.6% of those with STDs. Those with STDs (22.8%) are not significantly more likely than are those without STDs (19.0%) to be racial bridges. They are, however, significantly more likely to have core status (17.5% versus 4.3%) and significantly less likely to have periphery status (57.4% versus 85.4%). These preliminary results suggest that sexual network factors are potential determinants of racial and gender differences in the likelihood of having STDs.

Table 2.1 also shows that those with STDs and those without STDs differ on some but not all sociodemographic characteristics. For example, those with STDs do not differ systematically from those without STDs in terms of age. However, those with STDs generally do have lower levels of education and lower income. They are also less likely to be married.

In terms of sexual behavior, those with STDs are much more likely to have initiated sexual intercourse before age 16. They are more likely to report same sex intercourse, and they are slightly more likely not to have used a condom. Those with STDs are also disproportionately likely to report that they use illicit drugs (39.2% versus 21.2%) and engage in anal sex (52.9% versus 34.5%).

Do social locations differ in their tendencies to report having an STD? Figure 2.1 presents the percentage of respondents with STDs by social location. It shows that 5.54% of Black females reported having an STD. In comparison, 1.23% of White females, 1.15% of Latina females, and 2.12% of other race females report having an STD. It also shows that 4.37%

of Black males, 1.14% of White males, .91% of Latino males, and .87% of other race males report having an STD. Generally, these results suggest that there are significant racial and gender differences in the likelihood of having an STD, as these results produce a  $\chi^2$  value of 244.3 with 7 degrees of freedom and a probability value of  $p < .001$ .

A central point of focus for this chapter is sexual network factors. Are sexual network factors associated with the likelihood of reporting an STD? Figure 2.2 presents the percentage of respondents with STDs by Sexual Network factors. Overall, 1.69% of respondents have STDs. Consistent with Hypothesis 1A, Figure 2.2 shows that those with concurrent partners (6.17%) have higher rates of STDs than do others. It also shows that consistent with Hypothesis 2A, those with risky partners have higher rates of STDs (12.75%) than do others. It also shows that 6.5% of core members have STDs in comparison with 1.14% of periphery members. The chart shows that 2.02% of racial bridges have STDs. These results suggest that sexual network factors can potentially help explain racial disparities in STDs.

Do sexual network factors help account for the gaps in reporting STDs by social location? To address this question, multivariate analysis is appropriate. Because the dependent variable—reporting an STD—is binary, the multivariate statistical analyses use the binary logit model with two mutually exclusive outcomes (reporting an STD or not reporting an STD). The relationship between the dependent variable and the independent variables should be nonlinear. According to Liao (1994: 12), it can be represented as a generalized linear model with logit link and binomial distribution:

$$\frac{P}{1-P} = e^{a + \beta_1 x_1 + \dots + \beta_k x_k}$$

where  $P$  is the probability of an event occurring, and  $1-P$  is the probability of that event not occurring. This model outcome is a linear function,  $e$ , of the predictors. Specifically, the log-odds of success (the logit of the probability) is fit to the predictors using linear regression, where  $\alpha$  is the intercept (the value of  $z$  when the value of all independent variables are zero) and  $\beta_1$  through  $\beta_K$  are logit coefficients of  $X_1$  through  $X_K$ , respectively Liao (1994). A positive coefficient means that the explanatory variable increases the probability of the outcome. A negative coefficient means that the variable decreases the probability of that outcome. A near-zero coefficient means that the risk factor has little influence on the probability of that outcome.

By taking the natural logarithm of this equation, it is possible to calculate odds ratios (for ease of interpretation when the independent variable of interest is categorical) (Liao, 1994).

$$\ln\left(\frac{P(Y=1)}{1-P(Y=1)}\right) = \alpha + \beta_1 x_1 + \dots + \beta_k x_k;$$

Model 1 of Table 2.2 presents the results from logistic regression that predicts the log odds and odds ratios of reporting an STD with social location. This first model examines only the relationship between social location and reporting an STD without controls for other factors. For ease of interpretation, the first column presents the results as log-odds coefficients; the second column presents these same results as odds ratios. Model 1 shows that the odds of White women reporting an STD are .212 times as high as the odds for black women reporting an STD. The odds for Latina women are .199 times as high, and the odds for other race women are .370 times as high as the odds for black women reporting an STD. Compared with Black women, Black men have odds that are .78 times as high, white men have odds that are .196 times as high, Latino males have odds that are .151 times as high, and other race men have odds of reporting an STD that are .156 times as high. The only social location that is not statistically significantly

different from Black women are Black men ( $p = .212$ ). Model 1 produces a goodness-of-fit test statistic of 0.0 with a p-value of 1.0. This suggests an excellent fit of the data.

How does the relationship between social location and STD reporting change when other factors are taken into consideration? Model 2 of Table 2.2 shows that when other factors such as education, income, age, immigrant status, marital status, age at first sex, participation in same sex intercourse, condom use, drug use, and participation in anal sex intercourse are taken into account, the differences in the tendency to report having STDs between African American women and other social locations becomes somewhat smaller except for other race women. For example, the odds ratio of White women reporting an STD compared with Black women reporting an STD increase from .212 to .251. Similarly, the odds ratio of Latina women reporting an STD compared with Black women reporting an STD increase from .199 to .247. The odds ratio of White men reporting an STD compared with Black women reporting an STD increase from .196 to .284. And the odds ratio of Latino men reporting an STD compared with Black women reporting an STD increase from .157 to .173. Yet, the differences between Black women and White women, Latina women, and Latino men remain statistically significant when these baseline factors are taken into account.

In addition to these remaining differences, the results in Model 2 suggest that for every unit increase in education by one year, the likelihood of reporting an STD decreases by .108. This relationship is statistically significant ( $p < .01$ ). For every unit increase in income by ten thousand dollars, the likelihood of reporting an STD decreases by .106 ( $p < .01$ ). For every unit increase in age by one year, the likelihood of reporting an STD decreases by .013. This relationship, however, is not statistically significant ( $p = .619$ ). The odds of reporting an STD do

not vary systematically by marital status. For every unit increase in age at first sex by one year, the likelihood of reporting an STD decreases by .230 ( $p < .05$ ). Compared with those who do not engage in same sex intercourse, the odds of reporting an STD among those who engage in same sex intercourse are almost 2.5 times greater ( $p < .001$ ). There are no systematic differences in reporting an STD by condom use or by drug use. And compared with those who do not engage in anal sex intercourse, the odds of reporting an STD among those who engage in anal sex are 1.59 times greater ( $p < .01$ ). Model 2 produces a goodness-of-fit test statistic of 0.32 with a p-value of .724. This suggests an excellent fit of the data.

What happens to the gap in the tendency to report STDs when sexual network factors are taken into account? There are several observations that can be made. Model 3 in Table 2.2 shows that when taking into consideration the baseline and sexual network factors, the STD gap between Black women and all other social locations except other race women becomes somewhat larger when baseline and sexual network factors are taken into consideration (i.e., in Model 3 versus Model 2). In particular, the odds of White women reporting an STD decrease from .251 to .247 times as high as the odds for black women reporting an STD ( $p < .001$ ). In other words, the STD gap between Black women and White women actually becomes somewhat larger when sexual network factors are also taken into consideration. Similarly, the STD gap between Black women and Latina women becomes somewhat larger (from .247 to .218) when baseline and sexual network factors are taken into consideration ( $p < .001$ ). The odds of other race women reporting an STD increase from .640 to .647 times as high as the odds for black women reporting an STD ( $p > .05$ ). The STD gap between Black women and Black men becomes somewhat larger as the odds decrease from .885 to .648 times as high as the odds for black

women reporting an STD ( $p > .05$ ). The STD gap between Black women and White men becomes somewhat larger as the odds decrease from .284 to .270 times as high as the odds for black women reporting an STD ( $p < .001$ ). The STD gap between Black women and Latino men becomes somewhat larger as the odds decrease from .173 to .137 times as high as the odds for black women reporting an STD ( $p < .001$ ). And the STD gap between Black women and other race men becomes somewhat larger as the odds decrease from .296 to .209 times as high as the odds for black women reporting an STD ( $p < .01$ ). In essence, sexual network factors do not explain the social location differences in STDs.

Model 3 in Table 2.2 also provides evidence concerning Hypotheses 1-5. Consistent with the expectations of Hypothesis 1A, net of other factors, those who have partners in concurrent relationships are significantly more likely to report an STD than those without concurrent partners. The odds of those with partners in concurrent relationships reporting an STD are 1.868 times higher than are the odds for those without partners in concurrent relationships reporting an STD. This pattern is similar to what was presented in Figure 2.2.

What about the relationship between having risky partners and reporting an STD? Consistent with the expectations of Hypothesis 2A, net of other factors, the odds of those with risky partners reporting an STD are 2.666 times higher than are the odds for those without such risky partners reporting an STD. Having a risky partner significantly increases one's chances of reporting an STD ( $p < .01$ ).

Model 3 also shows, contrary to the expectations of Hypothesis 3A, those who are core members are no more likely than those who are not core members to report an STD. However, it shows that consistent with the expectations of Hypothesis 4A, those who are periphery members

are less likely than those who are not periphery members to report an STD, as the odds of those with periphery membership reporting an STD are .608 times as high as the odds for those without periphery membership. The number of sexual partners in one's network is somewhat predictive of reporting an STD.

Finally, Model 3 of Table 2.2 presents the relationship between racial bridging and reporting an STD. Consistent with Hypothesis 5A, net of other factors, those who engage in racial bridging are no more likely to have contracted an STD than are those who do not engage in racial bridging. Model 3 produces a goodness-of-fit test statistic of 1.30 with a p-value of .2727. This suggests a very good fit of the data.

But do sexual network factors work in different ways for different social locations as specified in Hypotheses 1-5? Table 2.3 provides some answers. Due to the number of interaction terms (35 in total), the table has been structured so that each column presents the interaction between social location and one of the sexual network factors while controlling for all other factors. For example, Model 1 provides results for those social location by concurrent partner status (e.g., White women with concurrent partners). Generally, interaction terms were included in the analysis because the sexual network literature suggests that the effects of sexual network factors vary for African American women and men versus other racial and gender groups. Goodness-of-fit tests suggest that all of the social location by sexual network interaction models provide good fit. Still, there is the more central question about whether the sexual network factors differ in their relationship to reporting an STD by social location. If the network factor differs in their relationship to reporting an STD by social location, the interaction terms will be statistically significant. In order to provide the reader with a more interpretable display of the

results, the results in Table 2.3 have been converted into predicted probabilities ( $\hat{p}$ ) and are reported in Table 2.4 and as graphs in Appendix D. The results reported in Model 1 of Table 2.4 are based on the logistic regression models that include the social location by sexual network interaction terms reported in Model 1 of Table 2.3. Similarly, results reported in Model 2-5 of Table 2.4 are also based on the logistic regression models that include the social location by sexual network interaction terms reported in Model 2-5 of Table 2.3.

Table 2.4 presents the predicted probabilities of reporting an STD by social location and sexual network factors (net of other factors). When an interaction term is statistically significant in Table 2.3, its corresponding predicted probability is highlighted (in bold) and with an asterisk (\*) in Table 2.4. There are four such significant interaction terms: (1) Latina women who are in concurrent relationships, (2) Latina women with risky partners, (3) Latina women with core memberships, and (4) other race men who racially bridge.

Recall that Hypothesis 1A predicts that those with concurrent partners will be more likely to report an STD. Hypothesis 1B predicts that, compared with African American women with concurrent partners, African American men with concurrent partners are less likely to report an STD, net of other factors. Moreover, sexual network theory leads to the expectation that, compared with African American women with concurrent partners, White women, White men, Latina women, Latino men, and other race women and men with concurrent partners are less likely to have an STD, net of other factors. Model 1 in Table 2.4 presents evidence concerning these expectations. It shows that net of baseline and other sexual network factors, compared with African American women with concurrent partners ( $\hat{p}=.064$ ), African American men with concurrent partners ( $\hat{p}=.0131$ ) are no less likely to report having an STD. Model 1 of this table



also shows that contrary to the expectations of sexual network theory, compared with African American women with partners in concurrent relationships, White women ( $\hat{p}=.0078$ ), other race women ( $\hat{p}=.0103$ ), White men ( $\hat{p}=.0086$ ), Latino men ( $\hat{p}=.0034$ ), and other race men with partners in concurrent relationships ( $\hat{p}=.006$ ) are no less likely to report an STD, net of other factors. In other words, even though concurrency is significantly related to tendency to report having an STD, its relationship to reporting an STD does not differ significantly for Blacks versus White women, other race women, White men, Latino men, nor other race men. Latina women, however, do differ from Black women in how concurrency is related to reporting an STD. Net of other factors, Latina women with concurrent partners reporting an STD ( $\hat{p}=.0043$ ) are significantly less likely than are Black women with concurrent partners reporting an STD ( $\hat{p}=.064$ ). Net of other risk factors, having a partner who is in concurrent relationships increases the likelihood of reporting an STD; still, race- and gender-differentiated patterns in the relationship between concurrency and reporting of an STD do little to account for the racial and gender gaps in reporting STDs.

The results in Model 2 of Table 2.3 provide support for Hypothesis 2A that having a risky partner increases the tendency to report having an STD. But net of other factors, are African Americans with high-risk partners significantly more likely to report having an STD compared with Whites with high-risk partners? Model 2 in Table 2.4 shows that, contrary to the expectations associated with Hypothesis 2B, African American women with high risk partners ( $\hat{p}=.06$ ) are not significantly more likely to report an STD compared with African American men ( $\hat{p}=.0125$ ) nor White women ( $\hat{p}=.0071$ ), other race women ( $\hat{p}=.0125$ ), White men ( $\hat{p}=.0074$ ), Latino men ( $\hat{p}=.0035$ ), nor other race men with high risk partners ( $\hat{p}=.0039$ ). Latina women with

high-risk partners ( $\hat{p}=.0041$ ) are significantly less likely than are their Black female counterparts to report an STD. The results suggest that with the exception of Latina women with risky partners, the risky partner-social location interaction terms are statistically non-significant. Yet, all main effects of social locations are significantly less likely to report having an STD than are Black women except for Black men and other race women (see Model 2 in Table 2.3). This suggests that even the social location by sexual network interaction terms did not explain the social location differences in reporting an STD.

Model 3 of Table 2.3 shows that net of other factors, core members are no more likely to report an STD than are non-core members. Contrary to Hypothesis 3B, Model 3 in Table 2.4 shows that African American women ( $\hat{p}=.041$ ) and men ( $\hat{p}=.009$ ) who are core members are no more likely to report an STD than are White women ( $\hat{p}=.0049$ ), other race women ( $\hat{p}=.0087$ ), White men ( $\hat{p}=.0047$ ), Latino men ( $\hat{p}=.0026$ ), and other race men who are core members ( $\hat{p}=.0033$ ). Latina women who are core members ( $\hat{p}=.0029$ ) are significantly less likely than are Black women to report an STD. Still, Black women generally are significantly more likely to report an STD than all social locations other than Black men and other race women even when the social location by core membership interactions are taken into account.

Model 4 of Table 2.3, however, shows that, net of other factors, the main effects of periphery membership remain statistically significant, as the odds of those with periphery membership reporting an STD are .508 times as high as the odds for those without periphery membership. This is consistent with Hypothesis 4A. Nevertheless, as Model 4 in Table 2.4 shows, African American women who are periphery members ( $\hat{p}=.0367$ ) are no more likely to report an STD than are any other social locations with periphery membership: White women

( $\hat{p}=.0037$ ), Latina women ( $\hat{p}=.0043$ ), other race women ( $\hat{p}=.0015$ ), Black men ( $\hat{p}=.0061$ ), White men ( $\hat{p}=.0042$ ), Latino men ( $\hat{p}=.0015$ ), and other race men who are periphery members ( $\hat{p}=.0005$ ). This is contrary to Hypothesis 4B. Still, Black women generally are significantly more likely to report an STD than all social locations other than Black men and other race women even when the social location by core membership interactions are taken into account.

Does racial bridging operate in the fashion predicted by Hypothesis 5A? Model 5 of Table 2.3 shows that, net of sociodemographic and behavioral factors, racial bridging tends to have little relationship to reporting an STD. This finding is consistent with Hypothesis 5A. Still, with the exception of other race males, these patterns are not differentiated by social location: White women ( $\hat{p}=.0055$ ), Latina women ( $\hat{p}=.0032$ ), other race women ( $\hat{p}=.0098$ ), Black men ( $\hat{p}=.0105$ ), White men ( $\hat{p}=.0053$ ), Latino men ( $\hat{p}=.0029$ ), and other race men who racially bridge ( $\hat{p}=.0037$ ). Even when taking such interactions into consideration, Black women are significantly more likely to report an STD than all social locations other than Black men. Thus, the patterns in the data are consistent with Hypothesis 5A but not Hypothesis 5B.

These results suggest that, net of sociodemographic and behavioral factors, the main effects of the sexual network factors are consistent with the predictions derived from sexual network theory (i.e., four out of five hypotheses). However, factors associated with sexual network theory do not appear to go very far in terms of explaining the racial and gender gaps in STDs. As pointed out above, sexual networks alone do not explain racial and gender disparities in STDs. Sexual network factors do not appear to be related to reporting STDs in ways that are systematically different for various social locations.

## **Discussion and Conclusions**

This chapter began with the observation that sexually transmitted diseases (STDs) represent a considerable burden to American society that disproportionately affects women and people of color. In particular, African Americans report substantially higher rates of chlamydia, gonorrhea, or syphilis than do whites (CDC, 2010). Generally, women report higher rates of these diseases than do men (CDC, 2010). The chapter put forth the idea that intersectionality offers a perspective that could be useful for understanding racial and gender disparities in reports of STDs. It also identified sexual network theory as a sociological explanation that could help account for differences in the likelihood of reporting STDs. In particular, sexual network theories of STDs posit that group-level dynamics such as having sex with people in concurrent partnerships, having sexual intercourse with risky partners, being involved in dissortative mating, and being involved in relationships across racial or other group lines account for differences in STDs.

These theories often imply a cultural pathology argument. They imply that if African Americans (and women) were in networks that were similar to whites (and men), their risks would be similar to those of whites (and men). In other words, if such group level differences are key, then after accounting for these sexual network factors, disparities among race by gender groups should disappear.

The chapter used nationally representative data from the National Survey of Family Growth to examine five hypotheses consistent with sexual network theory. The results from multivariate logistic regression analyses suggest that, net of sociodemographic and behavioral factors, the main effects of the sexual network factors are consistent with the predictions derived

from sexual network theory (i.e., four out of five hypotheses). However, factors associated with sexual network theory do not appear to go very far in terms of explaining the racial and gender gaps in STDs, as none of the sexual network by social location interactions were consistent with hypotheses derived from sexual network theory. Sexual network by social location interactions do little to explain racial and gender disparities in STDs. This was found to be true even when examining patterns only among those who had been screened for STDs within the past year.

Despite the relevance of sexual networks in explaining differences in STDs, they do not help explain much about the racial and gender gaps in STDs. Moreover, such theories do not usually provide a compelling explanation of why it is that particular kinds of people end up in the networks in which they are embedded. Although they assert that sexual segregation matters in maintaining racial disparities in STDs, they do not give sufficient attention to socioenvironmental factors (e.g., residential segregation) which may also be related to sexual segregation and related to racial disparities in STDs. In contrast, intersectionality calls for the examination of contextual factors. The next chapter will turn to such issues.

## **CHAPTER 3**

### **Separate but Unequal: Residential Segregation and Sexually Transmitted Diseases**

#### **Introduction**

In thinking about how to start this chapter on racial disparities in sexually transmitted diseases (STDs) at the community-level, I wanted to include some shocking statistics concerning the disparate impact of STDs on Black communities. For example, according to Newman and Berman (2008:88), more than 95% of black communities in the United States have rates of gonorrhea that exceed 100 cases per 100,000 residents. This contrasts with less than 1% of white communities. Such disparities are not limited to gonorrhea, as there are similar patterns for chlamydia (Newman and Berman, 2008). Community-level analysis suggests that these disparities are fueled by residential segregation and sexual network factors above and beyond individual level behaviors. However, as Greenberg (1990: 780) put it, “the poor ranking of America’s black population in the indices of good health is a scandal of such long standing that it has lost the power to shock.” I suspect, like other social ills such as mass incarceration and unemployment rates plaguing African Americans, it has become taken for granted that such inequalities are just the way things are.

Racialized patterns of disparities in disease are often of little concern to whites (Newman and Berman, 2008). Still, it is of the utmost importance for scholars to continue to examine those factors that create and sustain health disparities in order to keep these issues at the forefront of concern for the nation as a whole. For the most part, the health disparities literature suggests that high levels of residential segregation keep minority groups isolated, disadvantaged, and at risk for exposure to higher rates of violence, environmental toxins, lack of adequate health care,

incarceration, sex ratio imbalances, and concentrations of infectious diseases that increase risk for STDs (Massey and Denton, 1993; Acevedo-Garcia, 2000:1150; Williams and Collins, 2001; LaVeist et al., 2005; and LaVeist et al., 2011). Research evidence demonstrates that residential segregation (i.e., racial isolation) concentrates disadvantage for African Americans with respect to STDs. Missing from the research, however, is the impact that racial residential segregation has on those who live in white segregated areas. According to Beaulieu and Continelli (2011: 488), “there are theoretical reasons to believe that segregation not only negatively impacts black communities but that it also serves to benefit white communities.” Sociologists argue that whites utilize residential segregation to maintain social distance from African Americans in order to concentrate economic and social advantage (Massey and Denton, 1993; Bobo and Zubrinsky, 1996; Meyer, 2000; and Charles, 2003). It is plausible, therefore, that not only does residential segregation elevate rates of STDs among those in black segregated areas, but also that white isolation from blacks serves to provide residents of those areas with advantages that lower their rates of STDs.

The health disparities literature has called for additional examination of the link between residential segregation and STDs. The purpose of this chapter is to examine the link between residential segregation and STDs in various kinds of communities. This chapter examines the impacts of both black isolation and white isolation on STDs rates in communities with the lowest percentages of Black residents (less than .7%), intermediate percentages of Black residents (between .7% to 10.3%), and communities with the highest percentages of Black residents (more than 10.3%). Biello et al. (2012) and Pugsely et al. (2013) expanded the STD literature by exploring multiple dimensions of residential segregation. They did not, however, examine

whether advantages accrue to those in white-segregated communities in the form of lower STDs rates. Moreover, in order to understand how these dynamics operate in the national context, it would be preferable to look beyond metropolitan areas only. This research seeks to examine whether white isolation has the opposite effect by acting as a structural factor to concentrate advantage for those living in white-isolated areas.

This research uses a hypersegregation perspective to examine the case of STD disparities in communities in the United States (Massey and Denton, 1993). A hypersegregation perspective links key aspects of residential segregation with deleterious health outcomes (Massey and Denton, 1993). In doing so, it also utilizes an intersectional framework and uses county-level data to consider the link between residential segregation and chlamydia rates and gonorrhea rates in communities with the lowest percentages of Black residents, in communities with intermediate percentages of Black residents, and in communities with the highest percentages of Black residents. A central concern of the research is the independent relationship of residential segregation to STDs rates in various kinds of counties.

### **Hypersegregation Theory of Residential Segregation and STDs**

Racial residential segregation refers to “the degree to which two or more groups live separately from one another in different parts of the urban environment” (Massey and Denton, 1988: 282). Scholars suggest that residential segregation is a useful theoretical tool for the study of racial disparities in health outcomes such as STDs because it allows for the examination of structural inequality above and beyond individual- and group-level network factors (Biello et al., 2012: 1370). Such structural inequality can be thought of as racism, which operates through formal and informal policies and practices that segregate communities by race into dominant and



subdominant groups (Acevedo-Garcia, 2000: 1144-1145; Williams and Collins, 2001; LaVeist et al., 2011: 10).

In *American Apartheid*, Massey and Denton (1993) argue that African Americans continue to suffer high rates of poverty and other deleterious outcomes because of systemic residential segregation. They assert that American apartheid (i.e., hypersegregation) acts as a stratifying agent above and beyond individual level factors through “ongoing institutional arrangements and individual actions” rather than as a byproduct of a black middle class exodus or lack of access to jobs in the community (p. 143). They acknowledge that these factors impact black communities, but they assert that residential segregation serves as the “structural linchpin” to create and maintain black communities of concentrated disadvantage (p. 147). Their work demonstrates that, despite civil rights laws of the 1960s aimed at curbing discrimination in housing, African Americans remain residentially segregated from whites. They suggest that high levels of black isolation interact with other community-level characteristics such as low median socioeconomic status (SES) and sex ratio imbalances to exacerbate the effects of these characteristics and in turn concentrate disadvantage (p. 132). The American apartheid framework, therefore, is a structural theory that helps explain inequality in society because it links racial residential segregation to other community-level factors that act to create and maintain stratification outcomes in health, finances, housing, and politics between groups within shared geographic locations (p. 149).

Following the work of Massey and Denton (1993), Williams and Jackson (2005) argue that racial segregation is the leading cause of racial inequality in the U.S. because equity in housing represents a substantial portion of most people’s wealth. African Americans have been historically locked out of the housing market, especially at pivotal moments in history. This has

reduced their ability to amass wealth that could provide protective barriers against disease. Williams and Jackson (2005) argue that African Americans face ill effects in health at higher rates than whites because they suffer greater residential segregation than other minorities (Williams and Jackson, 2005: S20). This lack of wealth coupled with “where an individual lives—especially where one grows up—exerts a profound effect on one’s life chances” (LaVeist, 2005: 149). Such explanations help explain how where one lives affects their health even after controlling for socioeconomic status (LaVeist, 2005).

Despite the link between concentrated poverty and residential segregation, scholars note that residential segregation is not simply a mask for class segregation; rather, it is a result of racism that acts above and beyond socioeconomic factors (Jargowsky, 1996; Acevedo-Garcia 2000: 1145; Laviest, 2005: 177). Residential segregation has been found to be associated with numerous negative health outcomes (Massey and Denton, 2001; and Williams and Collins, 2001). Williams and Collins (2001) argue that residential segregation leads to racial differences in (1) neighborhood poverty, (2) access to neighborhood resources, and (3) exposure to violence. LaVeist (2005: 136-137) argues that health disparities can be partially explained by residential segregation because it predisposes minorities to worse health outcomes. He suggests that segregation keeps disadvantaged minority groups isolated and at risk for exposure to higher rates of violence, environmental toxins, lack of adequate health care, and incarceration.

Williams and Sternthal (2010) also argue that American apartheid is an institutional form of racism. It not only leads to differential outcomes in health for African Americans, but it serves to structure different communities in which African Americans find themselves. Williams and Sternthal (2010) also argue that residential segregation is associated with worse health. Because

African Americans face high levels of segregation, their health will be more strongly impacted by residential segregation than other groups (p. S20).

Hogben and Leichter (2008: S13) provide a review of several social determinants of STDs and argue that these determinants are related to STD disparities. They acknowledge that individual behaviors interact with specific characteristics of STD pathogens that influence contraction and transmission (p. S13). However, behavior alone does not account for racial disparities. To address this gap in the literature, they suggest that larger social forces such as residential segregation act as “an organizing principle that overlaps with other social determinants” and sexual networks to act as a leading cause in STD disparities (p. S14). They assert that segregation interacts with other social determinants and combines with “characteristics of the pathogen and broad societal norms and patterns of behavior to influence the epidemiology of infectious disease” (p. S14). These factors then influence the odds of contracting an STD. Although there are no easy solutions to eliminating STD disparities, Hogben and Leichter (2008) argue that interventions attempting to eliminate STD disparities must address residential segregation.

Hogben and Leichter (2008) put forth a reasonable argument that segregation is related to STD disparities. However, their argument is based on a literature review rather than first-hand empirical analysis. This research will extend their argument and test whether residential segregation matters for differential rates of STDs using national data.

According to Karlsen and Nazroo (2002: 624), the impacts of institutional discrimination— “discriminatory policies or practices embedded in organizational structures”— often seem invisible compared with overt individual acts of racism. They argue that racism in the

form of residential segregation operates as an invisible factor that helps to maintain the structural policies and practices that continue to locate African Americans in segregated communities, which expose them to higher rates of crime, unemployment, incarcerations, and diseases. Therefore, residential segregation must be addressed when attempting to examine and eliminate STD disparities.

Hogben and Leichter (2008: S13) see residential segregation as both a historical and present-day “manifestation of institutional racism.” This type of institutional racism acts as a force that pushes some groups into communities of risk, and it justifies an examination of residential segregation and its relationship to STD disparities from both a public health and social justice perspective. Similarly, despite policies outlawing housing discrimination and other efforts aimed at eliminating residential segregation, Williams and Jackson (2005: 328) suggest that residential segregation plays a role in maintaining racial disparities in health.

As LaVeist et al. (2011: 17) points out:

[r]acial segregation decreased somewhat between 2000 and 2010. For blacks living in cities with resident populations of 100,000 or more, segregation declined by about 6.6 percent and segregation for Hispanics decreased by 7.7 percent. Despite this relatively small decrease in segregation patterns, the U.S. remains highly segregated. For example, African Americans represent about 12.6 percent of the entire U.S. population. In turn, Hispanics represent some 16.3 percent of the population. Overall, there are approximately 38.9 million African-Americans and about 50.3 million Hispanics. To achieve full racial integration in American cities, nearly 57 percent of African Americans and 48 percent of Hispanics would have to move to different neighborhoods. In other words, 21.8million blacks and 24.2 million Hispanics would need to move, which is roughly equivalent to

relocating the entire population of the states of New York and Texas, respectively” (LaVeist et al., 2011:17).

These statistics highlight the continued residential segregation in the U.S. They caution, however, against the assumption that the concentration of African Americans creates these disparities; rather, building on Wilson (1981), they suggest that concentrated poverty and lack of resources creates deleterious living conditions to which African Americans are disproportionately exposed. There are also studies that suggest that when African Americans and whites reside in the same geographic areas with more equitable access to economic and healthcare resources, disparities in health outcomes decrease (Bleich et al., 2010; LaVeist et al., 2009; and Thorpe et al., 2008).

Residential segregation has been noted to have both a direct and an indirect effect on health disparities (Acevedo-Garcia 2000: 1150; LaVeist et al., 2005; LaVeist et al., 2011). For example, Acevedo-Garcia (2000) argues that residential segregation is directly related to infectious diseases disparities. Residential segregation facilitates the transmission of infectious diseases like tuberculosis because communities with elevated concentrations of infection and persons who are susceptible come into contact and increase the infection, which in turn increases the disparity in the general population. Scholars also argue that residential segregation’s role as a social organizing agent may indirectly help create and sustain social network interactions that can directly impact the spread of diseases by keeping certain groups isolated and concentrating infection by affecting the availability of partners (Ghani, Swinton, and Garnett, 1997; Acevedo-Garcia, 2000; Kaplan et al., 2009; and Biello et al., 2012).

According to the CDC (2006), residential segregation concentrates high rates of deleterious living conditions for minorities. These conditions expose residents, particularly youth who are extremely vulnerable to STDs. These youth are more likely to engage with other racially segregated partners who are more likely than those in other communities to be infected. Given the levels of isolation and the lack of adequate health care access, residential segregation in the form of black isolation helps to maintain and reinforce the disparities in STDs (CDC, 2006: 11).

Massey and Denton (1988) identify five dimensions of residential segregation: unevenness, isolation, concentration, clustering and centralization. These “[d]ifferent dimensions of residential segregation may have different effects on the wellbeing of minorities” (Acevedo-Garcia 2000: 1147). Although unevenness (i.e., dissimilarity), which measures the degree to which groups are separated, is a commonly used indicator of residential segregation in health disparities research, with respect to infectious diseases such as STDs, isolation is a more appropriate indicator (Acevedo-Garcia, 2000). Isolation “measures the extent to which a member of a racial or ethnic group is likely to be in contact with members of this same group (as opposed to members of other groups)” (Acevedo-Garcia 2000: 1147).

There are two central studies looking at the relationship between residential segregation and STDs (Biello et al 2012; Pugsly et al 2013). Using Surveillance data from 2003-2007 and Census data from 2000, Biello et al (2012) explored whether the five dimensions of residential segregation proposed by Massy and Denton (1988) were related to gonorrhea rates among African Americans. Biello et al (2012) found that three dimensions of residential segregation (i.e., isolation, dissimilarity, and centralization) were related to gonorrhea rates. Building on the Acevedo-Garcia (2000) model of residential segregation and TB, that research suggested that the

direct impact of residential segregation in the form of isolation on racial disparities in STDs may be a result of organizing communities into places where sexual networks are isolated, concentrated with infection, and have reduced access to sexual health resources.

Pugsely et al. (2013) examined the role of black isolation and the Gini index on average gonorrhea rates for 2005-2009 for 277 U.S. metropolitan statistical areas (MSAs). They found that those MSAs with high levels of black isolation had odds that were 3 times higher for having high gonorrhea rates than MSAs with low levels of black isolation. They did not find a significant relationship for income inequality and STDs rates. Pugsely et al. (2013) expanded the STDs literature by exploring multiple dimensions of residential segregation; however, it failed to examine whether advantages accrue to those in white-segregated communities in the form of lower STDs rates.

According to Anderson and Massey (2004:338) “segregation persists in the United States because whites benefit from it.” Missing from the research on STDs and residential segregation, however, is the impact that racial residential segregation has on those who live in white segregated areas. It is plausible that not only does residential segregation elevate rates of STDs among those in black segregated areas through concentrated disadvantage, but also that white isolation serves to provide residents of those areas with advantages that lower their rates of STDs. This research seeks to examine whether white isolation has a different relationship to STDs rates by acting as a structural factor to concentrate advantage for those living in white-isolated areas. The current analysis examines the link between residential segregation (both black isolation and white isolation) and chlamydia rates and gonorrhea rates in communities with the

lowest percentages of Black residents, in communities with intermediate percentages of Black residents, and in communities with the highest percentages of Black residents.

Based on the *American Apartheid* hypersegregation theory, I derive the following hypotheses:

*Hypothesis 6:* Net of other factors, black isolation is associated with increases in chlamydia and gonorrhea rates, but white isolation is associated with lower chlamydia and gonorrhea rates.

*Hypothesis 7:* In white counties, both black isolation and white isolation are associated with decreases in chlamydia and gonorrhea rates.

*Hypothesis 8:* In integrated and in disproportionately black counties, black isolation is associated with higher rates of chlamydia and gonorrhea, but white isolation is associated with lower rates of chlamydia and gonorrhea.

### **Socioenvironmental Factors and Disparities in STDs**

Socioenvironmental explanations offer a theoretical framework that attempts to explain racial disparities in health outcomes generally and STDs in particular. These explanations suggest that the communities in which individuals find themselves are related to health outcomes (Diez-Rioux, 2007; Thomas et al., 2010). Communities become sites for differential exposure to resources and risk factors that mediate transmission and treatment of STDs and help shape social interactions that may increase risk of transmission (LaVeist, 2005; Massey and Denton, 1993; Upchurch et al., 1999; Adlar, 2006). The community can be defined as “a group of people who share some or all of the following: geographic boundaries; a sense of membership; culture and language; common norms, interest, or values; and common health risk or conditions” (National



Research Council 2003:178-179). Community-level factors such as residential segregation and unemployment rates have increasingly become associated with health outcomes for individuals (Diez-Rioux, 2007; Beltran et al., 2011). Diez-Rioux (2007: 1) claims that “because place of residence is strongly patterned by social position, neighborhood characteristics could be important contributors to health inequalities by socioeconomic indicators or race/ethnicity.” By examining whether these community- contextual factors are related to disparities in STDs, I am being faithful to the tenets of intersectionality (Zerai and Banks, 2002). Below, this chapter reviews community-level factors at the community level (i.e., census tract, neighborhood, and county-level) that have been associated with disparities in health outcomes.

### **Sex Ratios**

Many scholars argue that mass incarceration policies place communities at risk for sex ratio imbalances and increased STDs (Adimora et al., 2009). Because of mass incarceration, residents face unstable relationships that become interrupted by bouts of imprisonment. This leaves women behind to form new sexual relationships and increases their number of lifetime partners, particularly with men who have multiple partners because of the sex ratio imbalance in the community, which is related to STDs (Adimora and Schoenbach, 2005). The literature suggests sex ratio imbalances influence sexual network practices by pushing residents into relationships where concurrent partnerships are tolerated by women because they feel as if there are few options (Aral, 1996; Valentino, 2008: S26). These concurrent partnerships can increase the risk of spreading infections more rapidly within the community ( Adimora and Schoenbach, 2010). Given the relationship between sex ratios and STDs, sex ratio will be included as a baseline factor when examining residential segregation and STDs rates.

**Population Density**

Scholars argue that where people live has great impact on their health. In particular, “the urban health penalty—the greater prevalence of a large number of health problems and risk factors in cities than in suburbs and rural areas” elevates rates of poor health (Leviton et al, 2000: 863). The research also suggests that there are deleterious effects of living in urban areas because increased density often results in higher rates of toxic environments, crime, and mortality (Williams and Collins, 2001). Urban areas often provide fewer restrictions on engaging in non-normative sexual behaviors (Aldrich, 2004). The lack of restriction may lead to elevated rates of STDs. Therefore, this analysis will include population density as a control variable in the baseline model.

**Immigrants**

The literature on health disparities suggests that immigrants typically have better health outcomes than do native-born citizens (National Research Council and Institute of Medicine 2013; LaVeist, 2005). Nevertheless, when it comes to infectious diseases, some immigrants have higher levels than their native-born counterparts. Assimilation theories suggest that as immigrants assimilate their health outcomes should become more similar to those in the host society (Abraido-Lanza et al., 2004). With respect to STDs, because those communities with higher rates of immigrants might have lower rates of STDs, this analysis will control for percent immigrants in the community.

**Latinos**

Despite facing economic barriers to health care, Latinos have better health outcomes than do groups with higher average incomes such as non-Hispanic whites (LaViest, 2005). This

tendency for Latinos to be healthier than non-Hispanic whites despite having lower socioeconomic status has become known as “the Latino paradox.” The Latino paradox is a puzzle that has confounded researchers for more than two decades. Several different hypotheses have been put forth to account for the observed health disparities in favor of Latinos (Zsembik and Fennell, 2005). Kaplan et al. (2009:108) looked at the relationship between racial/ethnic homogeneity and STDs in 77 communities in Chicago. They found that those “communities where 60% of the residents were Hispanic, rates of STDs were lower compared with neighborhoods where 60% of the residents were African American”. They argue that Latino communities may have lower rates because of the sexual mixing patterns that keep Latinos in sexually isolated pools from African Americans and because African American communities may have higher concentrations of STDs. This research calls for the inclusion of the percentage of Latinos in the community when examining the relationship between community level factors and STDs. Therefore, this analysis will include percentage Latino residents in the baseline model.

### **Income Inequality**

Income inequality is associated with negative health outcomes (National Research Council and Institute of Medicine, 2013; Farley, 2006). However, scholars disagree about the extent to which income inequality matters above and beyond neighborhood income. Some scholars believe there is a direct impact of income inequality on health outcomes because it may reduce social cohesion (Daniels et al., 2000). Others suggest that communities with highly unequal income distributions are less able to devote neighborhood resources that promote good outcomes (Daly et al., 1998). Still others argue that there is no independent effect of income

inequality; rather, it could be masking those community level factors that are related to both income inequality and health outcomes (Lynch et al., 2004). Given the debates about the relationship between negative health outcomes and income inequality as an independent factor above and beyond median income, studies examining STDs and community level factors should take income inequality into account. Therefore, this analysis will include income inequality as one of the baseline factors in the segregation model.

### **Unemployment**

Unemployment status is related to health outcomes because many without jobs lack health insurance (National Research Council and Institute of Medicine 2013:5). Lack of insurance can reduce the likelihood of seeking treatment and testing for STDs, especially when individuals are asymptomatic. The lack of testing and treatment in a community may be linked with increasing concentrations of infection within communities. High unemployment has also been linked to the deterioration of communities, that can lead to increased drug sales and use, and the lack of social capital (Farley, 2006: S62). Unemployment may be linked with lower marriage rates, and lower marriage rates have been linked with multiple sexual partnerships, more lifetime partners, and STDs (Thomas and Thomas, 1999).

### **Healthcare Shortage**

The literature points to several neighborhood level factors that are related to STDs. Some scholars argue that community socioeconomic disadvantage is related to the ability to access quality healthcare and information about healthcare facilities in neighborhoods (Browning and Cagney, 2006). Lack of access to quality healthcare may reduce testing and treatment of individuals infected with STDs. This may increase the concentration of infection within a

community because individuals remain untreated and pose a risk to potential uninfected partners in their communities (Kaplan et al., 2009). Paradoxically, those neighborhoods with health care shortages may also have lower rates of reported STDs because fewer individuals are tested which could lead to fewer reports within those communities. Therefore, the “actual rates” could be distorted by the lack of reporting. Because healthcare shortages have been associated with higher levels of STDs (Kilmarx et al., 1997), this analysis will include health care shortage as a control variable in the residential segregation and STDs rates models.

### **SES: Income and Education**

Scholars argue that SES in the form of education and income are major drivers of racial disparities in health outcomes (Williams and Collins, 1995). Research suggests that low levels of socioeconomic status (SES) (i.e., education and income) are related to higher rates of infectious disease (Aral et al., 2005). “In the case of communicable diseases, the behaviors that expose individuals to infectious agents and, hence, the infectious agents themselves tend to cluster at lower levels of the social hierarchy. Both the practice of unsafe sex and the prevalence of all STIs tend to be greater among the poorer and less-educated subgroups” (Aral et al, 2005: S3). Du et al. (2009) in their longitudinal study of community-level factors and gonorrhea, found that lower levels of community SES were related to higher levels of gonorrhea. The literature also suggests that education is correlated with health outcomes (National Research Council and Institute of Medicine 2013: 165). Education is often a requirement for good jobs, high wages, and job benefits. Education has been linked with higher self-efficacy, ability to solve problems, and making better choices with respect to health behaviors (National Research Council and Institute of Medicine, 2013). Communities with higher rates of educated residents may have

lower unemployment rates, and better jobs and better benefits which may increase treatment of STDs within the community (Phelan et al., 2010). This analysis will include education as a control variable in the baseline model. Because research suggests that there is a relationship between neighborhood level SES and STDs in general, this analysis will include community-level income and education as part of the baseline model.

## **Data and Methods**

### **Data**

The analysis includes all U.S. counties that reported data on chlamydia infections, gonorrhea infections, and racial composition (N=3089). It relies on indicators from various sources measured at the county level, including the Centers for Disease Control (2009 STD Surveillance System and the Bridged-Race Population Estimates), the U.S. Census Bureau (Counties Data Files, 2005-2007 and 2006-2010 American Community Surveys, and the Small Area Income and Poverty Estimates Program), and the Health Indicators Warehouse. These indicators have been compiled into a single dataset in which county is the unit of analysis. Indicators include the rate of gonorrhea infection per 100,000, the rate of chlamydia infection per 100,000 and residential segregation (i.e., the black isolation index and the white isolation index).

The analysis relies on county-level data for several reasons. As McLaughlin and Stokes (2002: 100) suggest, although

neighborhoods may be important in metropolitan counties, nonmetropolitan residents are more likely to view the county as an important economic and social unit. The availability and accessibility of health care and of educational, civic, cultural, job, environmental, and recreational opportunities are largely determined at the local level and influenced by local structures. The county is often the

decision-making unit for providing and organizing local services (McLaughlin and Stokes, 2002: 100).

Moreover, in order to understand how these dynamics operate nationwide, it is preferable to look beyond metropolitan areas only. Using county-level indicators provides the ability to carry out nationwide analysis. For analysis purposes, “all parishes, boroughs and census area county-equivalents are considered counties ... all independent cities have been consolidated into (joined to) the broader surrounding geographic county” (Rand, 2011: 6). Below, the operationalizations of the variables used in the analysis are included.

### **Operationalizations**

Chlamydia rate refers to the number of reported chlamydia cases in the county per 100,000 residents. Reported cases from 2007, 2008, 2009, and 2010 were summed and averaged.

Gonorrhea rate refers to the number of reported gonorrhea cases in the county per 100,000 residents. Reported cases from 2007, 2008, 2009, and 2010 were summed and averaged.

This chapter uses the isolation index as an indicator of residential segregation because it is the most appropriate measure to use when studying infectious diseases (Acevedo-Garcia, 2000; and Pugsley, 2013). “Isolation measures the extent to which a member of a racial or ethnic group is likely to be in contact with members of the same group (as opposed to members of other groups)” (Acevedo-Garcia, 2000: 1154). Black isolation, therefore, is the extent to which blacks are likely to be in contact with other blacks as opposed to whites and other racial groups.

Similarly, white isolation is the extent to which whites are likely to be in contact with other whites as opposed to other racial groups. Research has shown that high black isolation (i.e., greater than 60 on a scale from 0 to 100) is related to the concentration of disadvantages such as

concentration of disease, crime, and unemployment (Massey and Denton, 1993; Bobo and Zubrinsky, 1996; Meyer, 2000; and Charles-Zubrinsky, 2003). White isolation, on the other hand, is associated with beneficial outcomes in health, wealth, and political power by isolating whites from communities with concentrated disadvantage (Beaulieu and Continelli, 2011).

In addition, counties were coded to indicate their racial composition. “Race of county” is a heuristic device used to categorize counties according to their percentage of black residents. Following the work of Benjamins et al. (2004), for each county, the percentage black residents was determined and coded to indicate whether that percentage was in the bottom quartile (i.e., less than .7% black residents), the middle two quartiles (i.e., between .7% and 10.3% black residents), or the top quartile (i.e., more than 10.3% black residents). For the convenience of the reader, these quartiles are referred to as counties with the lowest percentages of Black residents, counties with intermediate percentages of Black residents, and in counties with the highest percentages of Black residents.

Percent Latino. For each county, the percentage of residents who are Latino or Hispanic.

Sex Ratio. For each county, the number of male residents per 100 female residents.

Unemployment Rate. People are classified as unemployed if they do not have a job, have actively looked for work in the prior four weeks, and are currently available for work. The percent unemployed is the number of 16 year-old and over residents in the county per 100 individuals who are 16 year-old and over residents in the county.

Percent Immigrant. For each county, the percentage of residents who were not born in the United States.



Median Income. For each county, the median income is the dollar amount that divides the income distribution into two equal groups such that half of the population has income above that amount and of the population has income below that amount.

Shortage of Health Professionals. A county is designated as having a shortage of health professionals when it is an urban or rural area that has “a population to full-time equivalent primary care physician ratio ... greater than 3,000:1 and have unusually high needs for primary care services or insufficient capacity of existing primary care providers ... [or that] demonstrate that primary medical professionals ... are overutilized, excessively distant, or inaccessible to the population under consideration.” (Taylor, 2004:11). Counties that were designated as having a shortage of health professionals were coded 1, and others were coded 0.

Population Density. For each county, the number of residents per square mile.

Percent College Graduates. For each county, the percentage of residents 25 years old or older who have a college degree.

Income Inequality (i.e., Gini Coefficient). The Gini coefficient is a measure of statistical dispersion that measures the inequality among values of household income in a county. It is computed in such a way that a value of 0 indicates perfect income equality such that all values are the same and everyone has an exactly equal income and a value of 100 indicates maximum inequality such that one family has all the income inequality.

### **Analysis Strategy**

Stata 12.0 was used to carry out the data analysis and data management. The analysis is based on a series of Ordinary Least Squares (OLS) regression models in which county rates of chlamydia and gonorrhea are the dependent variables. The central independent variables are black isolation and white isolation. In addition, the models take into consideration college

graduation rates, percent Latino residents, sex ratios, unemployment rates, median income, percent immigrant residents, population density, and income inequality (i.e., the Gini index) as predictors of chlamydia rates and gonorrhea rates. The analysis also examines how these factors are related to chlamydia rates and gonorrhea rates when stratified by racial composition of county. The stratified analyses provide some assessment of how great the disparities in STDs are between counties with the lowest percentages of Black residents and counties with the highest percentages of Black residents, net of other county health-related factors.

Several diagnostics were performed in order to test for multicollinearity, homoscedasticity, and model specification. To test for multicollinearity, I used two diagnostic tests. Using the Stata `correlate` command to examine Pearson product-moment correlation coefficients for all pairs of variables used in the analyses (i.e., a correlation matrix). Generally, a correlation matrix indicates whether the independent variables to be used in the analyses are not too highly correlated so that they create problems of collinearity (Kleinbaum et al., 1987). Correlations greater than  $\pm .5$  may be problematic inasmuch as they may lead to unreliable estimates. When conducting multivariate analyses, I used the Stata variance inflation factor command (VIF) to scrutinize the correlations of the independent variables in greater detail. When values on this test exceeds 10.0, the correlations among the independent variables may be too great, and thus, indicate unreliable estimates (Institute for Digital Research and Education, 2012).

An assumption of OLS Regression is that of homoscedasticity—i.e., that the variance of errors is the same across all levels the dependent variable. In order to test for heteroscedasticity, (i.e., the violation of the assumption of homoscedasticity), I use the Breusch-Pagan test for

heteroscedasticity. These tests assess conditional heteroscedasticity, and it suggests that there may be problems with conditional heteroscedasticity when the chi-square value for the test has a probability value less than .05. When this occurs, either the model under consideration should be re-specified or it should be fit using robust standard errors (Berry and Feldman, 1985). Because all of the models run indicated the possibility of heteroscedasticity, the regressions were run using robust standard errors to correct for threats to stability and reliability (Berry and Feldman, 1985).

Model specification is assessed with a variety of test using the Stata command fitstat. Scott and Freese (2001:82) suggest that for all models, “fitstat reports the log-likelihoods of the full and intercept-only models, the deviance (D), the likelihood ratio chi-square (G2), Akaike’s Information Criterion (AIC), AIC\*N, the Bayesian Information Criterion (BIC), and BIC’.” They suggest that “fitstat is particularly useful for comparing two models.” They say that

“Akaike’s (1973) information criteria is defined as 
$$AIC = \frac{-2 \ln \hat{L}(M_k) + 2P}{N}$$
, where  $\hat{L}(M_k)$  is the likelihood of the model and  $P$  is the number of parameters in the model .... All else being equal, the model with the smaller AIC is considered the better fitting model” (Scott and Freese, 2001: 86).

In contrast, Scott and Freese (2001: 86) suggest that the Bayesian information criterion or BIC is “a measure of overall fit and a means to compare nested and non-nested models. ... BIC is defined as 
$$BIC_k = D(M_k) - df_k \ln N$$
 where  $df_k$  is the degrees of freedom associated with the deviance. The more negative the  $BIC_k$ , the better the fit.”

Stata's `fitstat` command also produces an adjusted coefficient of determination ( $R^2$ ) statistic, which offers an indication of model fit. According to Scott and Freese (2001:84) The  $R^2$  can be defined as:

$$R^2 = 1 - \frac{\sum_{i=1}^N (y_i - \hat{y}_i)^2}{\sum_{i=1}^N (y_i - \bar{y})^2} = \frac{\widehat{\text{Var}}(\hat{y})}{\widehat{\text{Var}}(\hat{y}) + \widehat{\text{Var}}(\varepsilon)} = 1 - \left[ \frac{L(M_{\text{Intercept}})}{L(M_{\text{Full}})} \right]^{2/N}$$

Similarly, the “adjusted  $R^2$ ” is defined as:

$$\overline{R}^2 = \left( R^2 - \frac{K}{N-1} \right) \left( \frac{N-1}{N-K-1} \right)$$

where  $K$  is the number of independent variables (Scott and Freese: 2001:84). This statistic provides a measure of how well observed outcomes are estimated by the model. It offers an indication of the proportion of total variation of outcomes explained by the model. Generally, the larger the adjusted  $R^2$ , the better the model fit.

As mentioned above, the analysis is based on a series of OLS regression models in which county rates of chlamydia and gonorrhea are the dependent variables. The following OLS regression models are estimated:

3. Chlamydia Rate =  $\alpha + \beta_1 \text{Black Isolation} + \beta_2 \text{White Isolation} + \beta_3 \text{Percent College Graduates} + \beta_4 \text{Percent Latino} + \beta_5 \text{Sex Ratio} + \beta_6 \text{Percent Unemployed} + \beta_7 \text{Median Income} + \beta_8 \text{Percent Immigrant} + \beta_9 \text{Health Professional Shortage} + \beta_{10} \text{Population Density} + \beta_{11} \text{Gini Index} + \varepsilon$

This equation suggests that chlamydia rates are a function of black isolation, white isolation, percent college graduates, percent Latino residents, sex ratios, percent unemployed, median income, percent immigrant residents, whether there is a health professional shortage, population density, and income inequality.

$$4. \text{ Chlamydia Rate} = \alpha + \beta_1 * \text{Black Isolation} + \beta_2 * \text{White Isolation} + \beta_3 * \text{Percent College Graduates} + \beta_4 * \text{Percent Latino} + \beta_5 * \text{Sex Ratio} + \beta_6 * \text{Percent Unemployed} + \beta_7 * \text{Median Income} + \beta_8 * \text{Percent Immigrant} + \beta_9 * \text{Health Professional Shortage} + \beta_{10} * \text{Population Density} + \beta_{11} * \text{Gini Index} + \varepsilon \mid \text{Percent Black Residents}$$

This equation suggests that chlamydia rates in counties of various racial compositions are a function of black isolation, white isolation, percent college graduates, percent Latino residents, sex ratios, percent unemployed, median income, percent immigrant residents, whether there is a health professional shortage, population density, and income inequality.

$$5. \text{ Gonorrhea Rate} = \alpha + \beta_1 * \text{Black Isolation} + \beta_2 * \text{White Isolation} + \beta_3 * \text{Percent College Graduates} + \beta_4 * \text{Percent Latino} + \beta_5 * \text{Sex Ratio} + \beta_6 * \text{Percent Unemployed} + \beta_7 * \text{Median Income} + \beta_8 * \text{Percent Immigrant} + \beta_9 * \text{Health Professional Shortage} + \beta_{10} * \text{Population Density} + \beta_{11} * \text{Gini Index} + \varepsilon$$

This equation suggests that gonorrhea rates are a function of black isolation, white isolation, percent college graduates, percent Latino residents, sex ratios, percent unemployed, median income, percent immigrant residents, whether there is a health professional shortage, population density, and income inequality.

$$6. \text{ Gonorrhea Rate} = \alpha + \beta_1 * \text{Black Isolation} + \beta_2 * \text{White Isolation} + \beta_3 * \text{Percent College Graduates} + \beta_4 * \text{Percent Latino} + \beta_5 * \text{Sex Ratio} + \beta_6 * \text{Percent Unemployed} + \beta_7 * \text{Median Income} + \beta_8 * \text{Percent Immigrant} + \beta_9 * \text{Health Professional Shortage} + \beta_{10} * \text{Population Density} + \beta_{11} * \text{Gini Index} + \varepsilon \mid \text{Percent Black Residents}$$

This equation suggests that gonorrhea rates in counties of various racial compositions are a function of black isolation, white isolation, percent college graduates, percent Latino residents, sex ratios, percent unemployed, median income, percent immigrant residents, whether there is a health professional shortage, population density, and income inequality.

## Preliminary Diagnostics and Preliminary Results

As an initial check for problems of multicollinearity, the analysis examined the correlations among the variables used in the analysis. The correlation matrix (see Appendix E) shows the Pearson product-moment correlation coefficients for all pairs of variables used in the analyses. It shows that there are 3,089 observations when using listwise deletion (i.e., the entire observation is omitted from the estimation sample if any of the variables to be used in the analyses are missing for that observation) (Bruin, 2006). The correlation matrix shows that all but two correlations are less than .5. Generally, this suggests that the independent variables to be used in the analyses are not too highly correlated so that they create problems of collinearity (Kleinbaum et al., 1987). The correlations between the gini index and black isolation ( $r = .508$ ) and between percent Latino and white isolation ( $r = -.786$ ) are potentially problematic. These correlations, along with others, will be scrutinized in greater detail with variance inflation factor (VIF) analysis in the multivariate analysis.

Figure 3.1 presents the rates of chlamydia and gonorrhea per 100,000 residents by the racial composition of counties. It shows that the overall rates of chlamydia are 407.8 per 100,000 residents. It also shows that in counties with the lowest percentages of Black residents, the rate is 205.3 per 100,000. This contrasts to 300.5 per 100,000 in counties with intermediate percentages of Black residents, and 563.1 per 100,000 in counties with the highest percentages of Black residents. In other words, the chlamydia rate in counties with the highest percentages of Black residents is more than 2.7 times higher than the rate in counties with the lowest percentages of Black residents. Similarly, the chart shows that, for all counties, the gonorrhea rate is 99.0 per 100,000. In counties with the lowest percentages of Black residents, the rate is 14.6 per 100,000.

This contrasts to 48.4 per 100,000 in counties with intermediate percentages of Black residents, and 171.0 per 100,000 in counties with the highest percentages of Black residents. Rates of gonorrhea are more than 11.7 times higher in counties with the highest percentages of Black residents than in counties with the lowest percentages of Black residents.

Table 3.1 presents selected characteristics of counties. In the final column (“All Counties”), it presents selected characteristics for all counties. It again shows that, for all counties, the chlamydia rate is 407.8 per 100,000, and the overall gonorrhea rate is 99.0. It also shows that the average black isolation index score is 26.7, and the average white isolation index score is 77.4. Overall, 14.4% of counties are located in the Northeast, 26.1% are in the Midwest, 36.7% are in the South, and 22.9% are located in the West. The average percentage of college graduates is 28.1. The average percentage of Latinos is 15.7. On average, there are 96.7 men per 100 women. The average unemployment rate is 8.0 percent. The overall median income is \$52,313. The percentage immigrant is 13.3. It shows that 4.4 percent of counties have a health care professional shortage. The average population density is 223.3 people per square mile. The average Gini index score is 44.4.

Table 3.1 also shows that, based on a difference of means test, counties differ by selected characteristics. For example, counties vary in their levels of black isolation. On average, counties with the lowest percentages of Black residents have black isolation scores of .7. This compares with 12.4 for counties with intermediate percentages of Black residents and 47.94 in counties with the highest percentages of Black residents. In contrast, counties with the lowest percentages of Black residents average white isolation index scores of 83.6. This compares with

80.3 for counties with intermediate percentages of Black residents, and 73.1 for counties with the highest percentages of Black residents.

A central concern of this analysis is whether counties with different racial distributions vary in their rates of chlamydia and gonorrhea. It is, therefore, appropriate to determine whether counties of different racial compositions differ on other characteristics that might be related to chlamydia rates and gonorrhea rates. Counties with the lowest percentages of Black residents are disproportionately located in the Midwest and West. Those counties with intermediary levels of Black residents are overrepresented in the West. Counties that have the highest percentages of Black residents are disproportionately located in the South. Counties with the lowest percentages of Black residents generally have the lowest percentage of college graduates, as 18% of residents of these counties hold college degrees in comparison with 22.8% of residents in counties with intermediate percentages of Black residents, and 21.1% of residents in counties with the highest percentages of Black residents. The table shows that 15% of residents of counties with the lowest percentages of Black residents are Latinos. This compares with 16.8% of residents in counties with intermediate percentages of Black residents, and 14.3% of residents in counties with the highest percentages of Black residents. Counties with the lowest percentages of Black residents generally have higher male to female sex ratios (99 men per 100 women) than do counties with intermediate percentages of Black residents (98 men per 100 women) and counties with the highest percentages of Black residents (94.7 men per 100 women). In counties with the lowest percentages of Black residents, the unemployment rate is 7.4%. This compares with 7.5% in counties with intermediate percentages of Black residents, and 8.7% in counties with the highest percentages of Black residents. Counties with the lowest percentages of Black residents have



median incomes of \$42,105. This compares with \$54,920 in counties with intermediary percentages of Black residents and \$50,216 for counties with the highest percentages of Black residents. Counties with the lowest percentages of Black residents have 9.4% immigrants. This compares with 10.9% in counties with intermediary percentages of Black residents and 16.5% for counties with the highest percentages of Black residents. It also shows that 16.4% of counties with the lowest percentages of Black residents have health care professional shortages. This compares with 3.6% in counties with intermediary percentages of Black residents and 4.2% of counties with the highest percentages of Black residents. Counties with the lowest percentages of Black residents have population densities of 76.5 people per square mile. This compares with 257.1 people per square mile in counties with intermediary percentages of Black residents and 197.6 people per square mile in counties with percentages of Black residents, and lower levels of income inequality. These factors are statistically controlled in the analysis.

### **Results**

Is racial residential segregation related to county rates of chlamydia and gonorrhea? How are black isolation and white isolation related to rates of chlamydia and gonorrhea? Do these relationships vary by racial composition of county?

Table 3.2 presents ordinary least squares (OLS) regression models predicting chlamydia rates with black isolation and white isolation. Model 1 shows that as black isolation increases by one, chlamydia rates increase by 6.21 per 100,000 ( $p < .001$ ). In contrast, as white isolation increases by one unit, chlamydia rates decrease by 3.96 per 100,000 ( $p < .001$ ). Generally, like much previous literature, these results suggest that black isolation, as an indicator of concentrated disadvantage, is associated with increasing rates of STDs. However, the results also

show that white isolation operates in a distinctly different fashion such that it is associated with decreasing rates of STDs. These patterns are fully consistent with Hypothesis 6. Black isolation and white isolation account for more than 50% of the variance in chlamydia rates. The AIC of 13.0 (versus 13.8 for the null model) and the BIC of 15433.3 (versus 18135.7 for the null model) suggest that the model provides a better fit of the data. Moreover, the VIF of 1.1 suggests that the independent variables are not too highly correlated to raise issues of reliability.

Model 2 indicates that, in counties with the lowest percentages of Black residents, white isolation is negatively related to chlamydia rates. In this model, however, black isolation is not systematically related to chlamydia rates. These patterns are partially consistent with Hypothesis 6. In counties with the lowest percentages of Black residents, racial isolation accounts for 33% of the variance in chlamydia rates. The AIC and BIC show improvements over the null model, and the VIF shows that the independent variables are not too highly correlated.

Model 3 provides information about the relationship between racial isolation and chlamydia rates in counties with intermediate percentages of Black residents. In counties with intermediate percentages of Black residents, white isolation is negatively related to chlamydia rates. In this model, however, black isolation is positively associated with increases in chlamydia rates. These patterns are fully consistent with Hypothesis 6. In counties with intermediate percentages of Black residents, racial isolation accounts for 44% of the variance in chlamydia rates. The AIC and BIC show improvements over the null model, and the VIF shows that the independent variables are not too highly correlated.

Model 4 shows the relationship between chlamydia rates and black isolation and white isolation in counties with the highest percentages of Black residents. It shows that, in counties

with the highest percentages of Black residents, black isolation is associated with increases in chlamydia rates but white isolation is not systematically related to chlamydia rates. These patterns are partially consistent with Hypothesis 6. In counties with the highest percentages of Black residents, racial isolation accounts for 31% of the variance in chlamydia rates. The AIC and BIC show improvements over the null model, and the VIF shows that the independent variables are not too highly correlated.

The results in Table 3.2 suggest that residential segregation is related to county rates of chlamydia. White isolation is associated with lower chlamydia rates. Black isolation, in settings other than counties with the lowest percentages of Black residents, is associated with higher rates of chlamydia. For the most part, these patterns are consistent with Hypothesis 6.

What happens to these patterns when other factors are taken into account? Table 3.3 presents OLS regression models predicting chlamydia rates with black isolation and white isolation, net of other county factors. Model 1 shows that, net of white isolation, region, percent college graduates, percent Latino, sex ratio, percent unemployed, median income, percent immigrant, healthcare professional shortage designation, population density, and income inequality, as black isolation increases by one, chlamydia rates increase by 4.3 per 100,000 ( $p < .001$ ). In contrast, as white isolation increases by one unit, chlamydia rates decrease by 6.5 per 100,000 ( $p < .001$ ). Like much previous literature, these results suggest that black isolation, as an indicator of concentrated disadvantage, is associated with increasing rates of STDs. However, the results also show that white isolation operates in a distinctly different fashion such that it is associated with decreasing rates of chlamydia.

Model 1 also shows that other factors are related to chlamydia rates. In particular, it shows that net chlamydia rates are significantly higher in the West than in the South. It also shows that chlamydia rates increase when the percent Latino residents decreases, the unemployment rate increases, median income decreases, population density increases, and income inequality increases. Net of other factors, counties with health professional shortages have lower chlamydia rates. Combined, these factors account for more than 64% of the variance in chlamydia rates in counties. The AIC and BIC show improvements over the racial isolation-only model, and the VIF shows that the independent variables are not too highly correlated.

Model 2 of Table 3.3 shows that, net of other county-level factors, in counties with the lowest percentages of Black residents, black isolation operates in a fashion similar to white isolation. Both black isolation and white isolation are associated with decreases in chlamydia rates, net of other factors such as region, education, percent Latino, sex ratios, percent unemployed, median income, percent immigrant, health professional shortage, population density, and income inequality. These results suggest that, in the context of counties with the lowest percentages of Black residents, black isolation does not represent concentrated disadvantage. Indeed, several factors usually associated with concentrated disadvantage (e.g., lower education, higher unemployment, lower income, and greater income inequality) appear not to be related to chlamydia rates in counties with the lowest percentages of Black residents as they are in the general population. In counties with the lowest percentages of Black residents, there is no systematic relationship between education and chlamydia rates, unemployment and chlamydia rates, income and chlamydia rates, nor income inequality and chlamydia rates. These

findings are fully consistent with Hypothesis 6. These factors account for more than 65% of the variance in chlamydia rates in counties with the lowest percentages of Black residents.

Model 3 of Table 3.3 shows that, in counties with intermediate percentages of Black residents, net of other county factors, black isolation is associated with higher rates of chlamydia, but white isolation is associated with lower rates of chlamydia. In this context, the relationships between chlamydia rates and unemployment, median income, percentage immigrant residents, health professional shortages, and population density are also similar to those in the general analysis. Similarly, Model 4 shows that black isolation is associated with higher rates of chlamydia, but white isolation still associated with lower rates of chlamydia in counties with the highest percentages of Black residents. Again, factors such as percentage Latino residents, sex ratio, unemployment rate, median income, and income inequality are related to chlamydia rates in ways that are similar to the patterns for the general analysis. These results in Models 3 and 4 are fully consistent with Hypothesis 6, and they suggest that black isolation in the context of counties with intermediate percentages of Black residents and counties with the highest percentages of Black residents appears to be more consistent with the idea of concentrated disadvantage, as higher unemployment, and greater income inequality are related to chlamydia rates. In both Models 3 and 4, these factors account for more than 54% of the variance in chlamydia rates in counties with intermediary and the highest percentages of Black residents.

Table 3.4 presents OLS regression models predicting gonorrhea rates with black isolation and white isolation. Model 1 shows that as black isolation increases by one, gonorrhea rates increase by 3.1 per 100,000 ( $p < .001$ ). White isolation is not systematically related to gonorrhea rates ( $p = .278$ ). Similar to the case of chlamydia rates, black isolation and white isolation

operates in very different fashions such that black isolation is generally associated with increasing gonorrhea but white isolation is not. These patterns are partially consistent with Hypothesis 6. Black isolation and white isolation account for 59.9% of the variance in gonorrhea rates. The AIC of 11.0 (versus 12.0 for the null model) and the BIC of 9296.8 (versus 12424.2 for the null model) suggest that the model provides a better fit of the data. Moreover, the VIF of 1.1 suggests that the independent variables are not too highly correlated to raise issues of reliability.

Model 2 indicates that, in counties with the lowest percentages of Black residents, white isolation is negatively related to gonorrhea rates. In this model, however, black isolation is not systematically related to gonorrhea rates. These patterns are partially consistent with Hypothesis 6. In counties with the lowest percentages of Black residents, racial isolation accounts for 6% of the variance in gonorrhea rates. The AIC and BIC show slight improvements over the racial isolation-only model, and the VIF shows that the independent variables are not too highly correlated.

Model 3 provides information about the relationship between racial isolation and gonorrhea rates in counties with intermediate percentages of Black residents. In such counties, white isolation is negatively related to gonorrhea rates. In this model, however, black isolation is positively associated with increases in gonorrhea rates. These patterns are fully consistent with Hypothesis 6. In counties with intermediate percentages of Black residents, racial isolation accounts for 37% of the variance in gonorrhea rates. The AIC and BIC show improvements over the racial isolation-only model, and the VIF shows that the independent variables are not too highly correlated.

Model 4 shows the relationship between gonorrhea rates and black isolation and white isolation in counties with the highest percentages of Black residents. It shows that, in counties with the highest percentages of Black residents, black isolation is associated with increases in gonorrhea rates but white isolation is not systematically related to gonorrhea rates. These patterns are not fully consistent with Hypothesis 6. In counties with the highest percentages of Black residents, racial isolation accounts for 34% of the variance in gonorrhea rates. The AIC and BIC show improvements over the racial isolation-only model, and the VIF shows that the independent variables are not too highly correlated.

The results in Table 3.4 suggest that residential segregation is related to county rates of gonorrhea. White isolation is generally associated with lower gonorrhea rates. Black isolation, in settings other than counties with the lowest percentages of Black residents, is associated with higher rates of gonorrhea. For the most part, these patterns are consistent with Hypothesis 6.

Table 3.5 presents OLS regression models predicting gonorrhea rates with black isolation and white isolation, net of other county factors. Model 1 shows that, net of white isolation, percent college graduates, percent Latino, sex ratio, percent unemployed, median income, percent immigrant, healthcare professional shortage designation, population density, and income inequality, as black isolation increases by one, gonorrhea rates increase by 2.4 per 100,000 ( $p < .001$ ). In contrast, as white isolation increases by one unit, gonorrhea rates decrease by 1.8 per 100,000 ( $p < .001$ ). Again, these results suggest that black isolation, as an indicator of concentrated disadvantage, is associated with increasing rates of STDs. However, the results also show that white isolation operates in a distinctly different fashion such that it is associated with decreasing rates of gonorrhea.

Other factors are related to gonorrhea rates in ways similar to their relationship to chlamydia rates. In particular, it shows that as the percentage of college graduates increases, gonorrhea rates decrease ( $p < .05$ ). It also shows that gonorrhea rates increase when the percent Latino residents decreases, median income decreases, and population density increases. Net of other factors, counties with health professional shortages have lower gonorrhea rates. Combined, these factors account for more than 67% of the variance in gonorrhea rates in counties. The AIC and BIC show improvements over the racial isolation-only model, and the VIF shows that the independent variables are not too highly correlated.

Model 2 of Table 3.5 shows that, net of other county-level factors, in counties with the lowest percentages of Black residents, black isolation is not systematically related to gonorrhea rates ( $p = .064$ ). White isolation, however, is associated with decreases in gonorrhea rates ( $p < .05$ ), net of other factors such as education, percent Latino, sex ratios, percent unemployed, median income, percent immigrant, health professional shortage, population density, and income inequality. Again, these results suggest that, in the context of counties with the lowest percentages of Black residents, black isolation does not represent concentrated disadvantage; indeed, several factors usually associated with concentrated disadvantage (e.g., lower education, higher unemployment, and lower median income) appear not to be related to gonorrhea rates in counties with the lowest percentages of Black residents as they are in the general population. In counties with the lowest percentages of Black residents, there is no systematic relationship between education and gonorrhea rates, sex ratios and gonorrhea rates, unemployment and gonorrhea rates, nor median income and gonorrhea rates. These findings are mostly consistent with Hypothesis 6. These factors account for 27% of the variance in gonorrhea rates in counties



with the lowest percentages of Black residents. The AIC and BIC show improvements over the racial isolation-only model, and the VIF shows that the independent variables are not too highly correlated.

Model 3 shows that, in counties with intermediate percentages of Black residents, net of other county factors, black isolation is associated with higher rates of gonorrhea, but white isolation is still associated with lower rates of gonorrhea. In this context, the relationships between gonorrhea rates and unemployment, median income, percentage immigrant residents, health professional shortages, and population density are also similar to those in the general analysis. Similarly, Model 4 shows that black isolation is associated with higher rates of gonorrhea, but white isolation is not significantly associated with rates of gonorrhea in counties with the highest percentages of Black residents ( $p = .058$ ). Again, factors such as percentage Latino residents, sex ratio, unemployment rate, median income, and income inequality are related to gonorrhea rates in ways that are similar to the patterns for the general analysis. These results in Models 3 and 4 are mostly consistent with Hypothesis 6, and they suggest that black isolation in the context of counties with intermediate percentages of Black residents and counties with the highest percentages of Black residents appears to be more consistent with the idea of concentrated disadvantage, as higher unemployment and lower income inequality are associated with higher gonorrhea rates. In both Models 3 and 4, these factors account for more than 50% of the variance in gonorrhea rates in counties with intermediary and the highest percentages of Black residents. In both Models 3 and 4, the AIC and BIC show improvements over the racial isolation-only model, and the VIF shows that the independent variables are not too highly correlated.

The results of these analyses are very similar to those for chlamydia rates. In particular, in counties with the lowest percentages of Black residents, black isolation does not operate in a fashion similar to how it does in counties with the highest percentages of Black residents. White isolation is generally associated with decreases in gonorrhea rates, net of other factors. Models 3 and 4 show that, in counties with intermediary and the highest percentages of Black residents, net of other county factors, black isolation is associated with higher rates of gonorrhea, but white isolation is generally associated with lower rates of gonorrhea.

The chapter has examined several questions: Is racial residential segregation related to county rates of chlamydia and gonorrhea? How are black isolation and white isolation related to rates of chlamydia and gonorrhea? Do these relationships vary by race of county? Overall, these results suggest that racial residential segregation is related to county rates of chlamydia and gonorrhea. Generally, black isolation is associated with increasing rates of chlamydia and gonorrhea and white isolation is related to decreasing rates of chlamydia and gonorrhea. However, in counties with the lowest percentages of Black residents, both black isolation and white isolation are associated with decreases in chlamydia, net of other factors. The patterns for gonorrhea are a bit more ambiguous, but clearly in such counties, black isolation does not have the same relationship to gonorrhea rates as it does in counties with higher percentages of Black residents. In counties with intermediary and the highest percentages of Black residents, black isolation is associated with increases in chlamydia rates and gonorrhea rates.

Figure 3.2 presents predicted chlamydia rates and gonorrhea rates by racial composition of county if black isolation equaled 0. This chart shows that the overall rates of chlamydia would decline by more than 33% (down from 407.8 to 272.5). It also shows that rates in counties with

the highest percentages of Black residents would decline by more than 25% (down from 563.1 to 411.1). The chlamydia rates in counties with the lowest percentages of Black residents would increase by less than 3% (up from 205.3 to 210.7). However, chlamydia rates in counties with intermediate percentages of Black residents would fall by more than 20% (down from 300.5 to 235.1).

Figure 3.2 also shows that overall gonorrhea rates would decline by more than 62% (down from 99.0 to 36.9). It also shows that rates in counties with the highest percentages of Black residents would decrease by more than 55% (down from 171.0 to 76.2). The gonorrhea rates in counties with the lowest percentages of Black residents would increase by 49% (up from 14.6 to 21.8). However, the gonorrhea rates in counties with intermediate percentages of Black residents would fall by more than 47% (down from 48.4 to 25.6).

Another way of understanding these results is in terms of disparities in STDs rates by racial composition of county. Recall that in Figure 3.1 the chlamydia rate for counties with the highest percentages of Black residents was 563.1 per 100,000 residents and the chlamydia rate for counties with the lowest percentages of Black residents was 205.3 per 100,000 residents. This suggests a disparity of 357.8 cases or a rate that is more than 2.7 times higher in counties with the highest percentages of Black residents. If black isolation were reduced to 0, it is estimated that the chlamydia rates for counties with the highest percentages of Black residents would be 407.8 and the rates for counties with the lowest percentages of Black residents would be 210.7. This would yield a disparity of 197.1 cases or a rate that is 1.9 times higher in counties with the highest percentages of Black residents. In terms of gonorrhea, the rate for counties with the highest percentages of Black residents was 171.0 and the rate for counties with the lowest

percentages of Black residents was 14.6. This suggests a disparity of 156.4 cases or a rate that is more than 11.7 times higher in counties with the highest percentages of Black residents. If black isolation were reduced to 0, it is estimated that the gonorrhea rates for counties with the highest percentages of Black residents would be 76.2 and the rates for counties with the lowest percentages of Black residents would be 21.8. This would yield a disparity of 54.4 cases or a rate that is 3.5 times higher in counties with the highest percentages of Black residents. In other words, the black county-white county chlamydia and gonorrhea rates disparities would be greatly reduced if black isolation were eliminated.

These results illustrate how powerfully residential segregation is related to STDs rates. More specifically, they show the role of black isolation in perpetuating disparities in chlamydia rates and gonorrhea rates between counties with the lowest percentages of Black residents and counties with the highest percentages of Black residents.

### **Discussion and Conclusions**

Black isolation is an indicator of residential segregation that measures how likely blacks are to encounter other blacks. When black isolation is high, research suggests that blacks are less likely to have access to social, economic, and political resources that help sustain good health. In counties with the highest percentages of Black residents, the level of black isolation on average is 47.9. This suggests that counties with the highest percentages of Black residents have levels of black isolation that routinely approach high isolation. With respect to this concentrated disadvantage represented by black isolation, counties with the highest percentages of Black residents suffer from higher rates of STDs, net of other factors. In contrast, counties with the lowest percentages of Black residents have an average black isolation index of .7. This suggests

that the concentrated disadvantage reflected by high black isolation described in the literature is relatively absent in these counties. This may help explain why, as black isolation increases in these counties with the lowest percentages of Black residents, it does not operate as expected. Consequently, blacks in counties with the lowest percentages of Black residents—even those who are more isolated in virtually all white counties—are probably more likely to have greater access to county resources. They, like whites in these counties, are shielded from the harshest impacts of concentrated disadvantage. Therefore, the effects of black isolation within counties with the highest percentages of Black residents is not equivalent to black isolation in counties with the lowest percentages of Black residents because the levels of concentrated disadvantage are not similar.

According to Massey (1995: 1229), “segregation persists in the United States because whites benefit from it.” I hypothesized that black isolation would increase rates of STDs. This research lends support to earlier studies, which demonstrate that concentrated disadvantage through high black isolation drives higher STDs rates. But this research also adds to the literature by also showing that white isolation is associated with lower rates of STDs. However, when interpreting these results, one must remain mindful that white isolation is often aimed at concentrating and preserving advantages for whites. But reducing black isolation reduces chlamydia rates and gonorrhea rates, and concentrated disadvantage leads to increased rates of these STDs. Previous research has demonstrated that when African Americans have access to county health benefits similar to those for whites, their levels of negative health outcomes are dramatically reduced (Bleich et al., 2010; LaVeist, et al., 2009; and Thorpe et al., 2008). Indeed, counties with intermediate percentages of Black residents have fewer concentrated disadvantages

and lower rates of STDs than do counties with the highest percentages of Black residents. While segregation does help explain the disparities in county rates of STDs, this research will attempt to examine other structural factors such as incarceration that may also help explain differences in county rates of STDs. The next chapter will add lockup rates and reentry locations to the analysis.

## **CHAPTER 4**

### **Incarceration, Ex-Offender Reentry, and Racial Disparities in Sexually Transmitted Diseases in Communities**

#### **Introduction**

Since the 1980s, driven by policies such as the “War on Drugs,” prisons have “emerged as a powerful and often invisible institution that drives and shapes social inequality” (Wakefield and Uggen, 2010: 400; Golembeski et al, 2005). For the 50 years between 1920 and 1970, the United States consistently incarcerated about 100 per 100,000 of its residents (Western and Pettit, 2010:7). Since then, the U.S. has quickly become a world leader in the incarceration of its own people with rates of 762 per 100,000 by 2000 (Huling, 2002:180). In 2010, there were approximately 2.3 million people incarcerated in the U.S. (Glaze, 2011). The skyrocketing rates of incarceration are not reflected equally among all groups, and the impact of incarceration does not affect all groups equally. For example, Black men represent roughly 6% of the U.S. population, but they comprise half of the prison population (Golembeski et al., 2005).

Compared with the general public, those in prison have 4 to 10 times higher rates of infectious diseases such as gonorrhea and chlamydia (Golembeski et al 2005). Prisoners are at higher risk for infectious diseases such as STDs while incarcerated because of sexual relationships with partners who are also incarcerated, limited access to condoms, rape, and drug use. Thus, prisons serve as sites for the spread of STDs (Massoglia 2008b, Schnittker & John 2007).

According to Massoglia et al. (2013:149), “[v]irtually all inmates are eventually released from prison, and each year more than 700,000 released offenders join more than 16 million current or former felons already residing in neighborhoods across the country.” Of those who

have been released, about half will return within three years (Pettit and Lyons, 2009: 728). As rates of incarceration have increased, so have the numbers of those who have been formerly incarcerated who reenter communities. Subsequently, many return to communities infected with undiagnosed STDs but with few resources to obtain treatment. The cycle of incarceration, release, and reentry exacerbates inequality in health for those who have been incarcerated, their families, and their communities (Solomon, 2006).

Research has shown that the burden of STDs is not borne equally among communities (Chessen et al, 2012). For example, according to the CDC (2008:17) the distribution of gonorrhea at the county-level varies with 46% of counties reporting rates of 19 or less per 100,000, 36.2% of counties reporting rates of 19 to 100 per 100,000, and 22.3% reporting rates of 100 or more per 100,000. According to Newman and Berman (2008:S8), more than 95% of those communities with the highest proportions of Black people in the United States have rates of gonorrhea that exceed 100 cases per 100,000 residents. This contrasts with less than 1% of white communities. Such disparities are not limited to gonorrhea, as there are similar patterns for chlamydia (Newman and Berman, 2008:S8). Chapter 3 showed that these disparities are linked to residential segregation above and beyond community-level factors such as median income, income inequality, level of access to healthcare, etc. This chapter examines the relationship between incarceration, ex-offender reentry locations, and county-level chlamydia and gonorrhea rates.

Chapter 3 showed that community-level factors such as median income, residential segregation, and healthcare access are associated with disparities in STDs. Given the high rates of incarceration and reentry, ecological studies of STD disparities should also examine the



relationship between incarceration and reentry from prison in addition to other community-level correlates of STDs. Therefore, this chapter examines the relationship among incarceration, ex-offender reentry locations, and chlamydia and gonorrhea rates in U.S. counties, net of other community-level factors that are related to STD rates.

### **Incarceration, Communities, and STDs**

Sociologists argue that prisons can be viewed as social institutions that stratify by race and gender and serve a major role in creating and maintaining economic, health, political, and other social disparities (Pettit and Western, 2004; Wacquant 2010, Alexander, 2010; Wakefield and Uggen: 388). Due to the mass incarceration occurring within the United States, sociologists view prisons as institutions that function similar to the educational system and the family. Thus, they should be included in attempts to understand how and why certain groups receive society's resources and others are denied (Grusky, 2001:3 ).

#### **Mass Incarceration Framework**

According to Wacquant (2001), U.S. mass incarceration is another form of social control that has emerged in place of slavery and Jim Crow laws. Michelle Alexander (2010), in her highly acclaimed book, *The New Jim Crow: Mass Incarceration in the Age of Colorblindness*, argues that mass incarceration and the justice system have reconstructed the disenfranchisement of individuals of color. After given a conviction, African American and Latinos men in particular, are cast into second-class citizenship through their felon status. She asserts that once an individual has been labeled as a felon, he has about the same legal rights to housing, voting, serving on a jury, health care, and other social benefits as an African American had during the

Jim Crow era. The lack of access to social resources due to this second-class citizenship places ex-offenders at risk for a host of health problems, including STDs (Alexander, 2010).

Pettit and Western (2004) argue that because of mass incarceration policies, low-income African American males are facing a new alternative life course trajectory: prison. They suggest that individuals typically go through life in ordered steps, “moving from school to work, then to marriage, to establishing a home, and becoming a parent” (p. 154). Moving through each phase in a timely fashion typically results in positive outcomes for the individual. Unfortunately, not all individuals in society who want to progress to adulthood through these stages are able to do so. In fact, for African American males with low SES, this trajectory may shift during early adolescence when they leave high school and enter prison. Given the massive changes in drug laws and associated incarceration, many African American males with low SES who drop out of high school are no longer moving from school to work; rather, they are now moving from school to prison. This historical shift in society to mass incarceration has, in essence, re-ordered the trajectory that these men face in life (Pettit and Western, 2004:154). They found that:

imprisonment has become a common life event for recent birth cohorts of Black non-college men. Among Black male high school dropouts, the risk of imprisonment had increased to 60%, establishing incarceration as a normal stopping point on the route to midlife. Imprisonment now rivals or overshadows the frequency of military service and college graduation for recent cohorts of African American men (Pettit and Western, 2004: 164).

Although scholars debate the causes of incarceration, clearly the burden falls disproportionately on the backs of poor Black men. Such inequalities call for the examination of the causes and the impacts of incarceration.

Thomas and Torrone (2006:1762) argue that mass incarceration “is tantamount to ‘forced migration,’” contributing to imbalances in neighborhood gender ratios and resulting in the potential for community health effects such as STD rates. Thomas and Torrone (2006: 1762) used data from “1995 through 2002 on entries, releases, and state prison system populations from the North Carolina Department of Corrections” to examine if incarceration variables were related to STDs. They found that higher rates of incarceration were associated with increased rates of STDs. However, their sample was not nationally representative. Therefore, their results were not generalizable to the larger population. This dissertation contributes to the literature by using national data to examine the relationship between incarceration and racial disparities in STDs.

Thomas, Torrone, and Browning (2010:102) used data “from the 1995 Program on Human Development in Chicago Neighborhoods, the Chicago Health Department, and the Chicago Police Department” to examine if there is a relationship between incarceration rates and STDs. They found that there was a relationship between reported cases of gonorrhea and chlamydia and incarceration. They argued that this relationship may be mediated by the level of social control and economic disadvantage of the neighborhood. They suggested that incarceration may influence both social control and economic disadvantage while altering gender ratios that impact dating patterns. Their study suggests the need to examine socioenvironmental forces such as incarceration. However, because their data were not nationally representative, generalizations from their study were limited. Nevertheless, these studies suggest that incarceration and other community-level factors may be related to STDs and may help explain racial disparities in such diseases.

Massive imprisonment of African American men is related to negative health outcomes such as increased risk of STDs (Moore and Elkavich, 2008). Prisoners' higher rates of infection and lack of testing and treatment also affect the concentration of STDs within their sexual networks once they leave prison. According to Rodgers et al. (2012), when individuals become incarcerated they have limited access to safe sex options, their sexual practices and networks may shift, and heterosexual men are more likely to engage in same-sex intercourse. The loss of their former partners and sex with high-risk partners leads to increased risk of infection. This creates an environment ripe for STD infection.

### **The Role of Ex-Offender Reentry in Community Rates of STDs**

According to Thomas et al. (2010), the inequitable removal of young African American men from their community through mass imprisonment shifts resources to white rural counties. This happens because federal policies allow counties where prisoners are housed to collect the political and economic resources associated with increased population due to the presence of those detained in prisons. This reduces resources in the home communities of those incarcerated that could be used to prevent the spread of STDs through screening and testing.

Mass incarceration also has consequence of shifting sex ratios and affecting sexual network patterns both when men and women are incarcerated and reenter their communities (Thomas et al, 2010). Thus, mass incarceration also shifts sexual/dating patterns within communities. This suggests that incarceration plays a role in racial disparities above and beyond the individual level, thus I would expect . *Hypothesis 9:* As incarceration rates in counties increase, the rates of chlamydia and gonorrhea will increase.

Massoglia et al. (2008) analyzed data from the National Youth Survey to examine the relationship between incarceration, infections, and stress-related outcomes. They found that individuals who had been incarcerated were more likely to suffer from infectious diseases and stress-related illnesses compared with those who had not been incarcerated. They found that prisons affect individual health. They suggested that prisons, similar to neighborhoods in which individuals live, affect inmates' health. Given that those incarcerated are likely to cycle in and out of prison and other institutions of social control (such as half-way houses and jails), examining the impact of both incarceration and prison reentry locations on county rates of STDs is important.

Prisons are, in essence, “dangerous neighborhoods” that increase the risk of exposure to infectious diseases such as STDs; therefore, scholars need to consider prison exposure to be a social determinant of health (Massoglia, 2008). However, the effects of incarceration do not end upon release. “As more than 95% of incarcerated individuals eventually reenter the general community, amplification of infectious diseases during incarceration poses definite risks to the communities to which infected and untreated inmates return” (Awofeso, 2010: 27). When prisoners are released back into communities, they often face challenges to obtaining stable housing. They typically stay with relatives, in homeless shelters, or in residential reentry centers (RRCs) (i.e., halfway houses) (Bureau of Prisons, 2013). These halfway houses for ex-offenders are referred to as “reentry locations” within this chapter. Instability in housing, employment, mental health services, and exposure to violence, often lead to risky sexual behaviors and re-offending in order to make money. This helps create and maintain a cycle of incarceration-release-and incarceration (Bureau of Prisons, 2013).

When prisoners are going to be released, they begin the process of reentry. The Second Chance Act of 2007 is a federal law that provides assistance for prisoners being released. The Act provides funding for communities to provide housing, employment, and surveillance of formerly incarcerated individuals to aid in their reentry into the community (Bureau of Justice Assistance, U.S. Department of Justice, 2013). According to the Federal Bureau of Prisons (2013), these facilities function to provide a structured environment, reduce homelessness, and help with employment and other social services as individuals transition back into communities.

Reentry locations are intended to help released prisoners transition from incarceration to the community by providing a more structured environment than would be available if the prisoner was released directly to the community without such support. The facilities are typically run by corrections departments or community organizations that subcontract through the department of corrections. Although prisoners are eligible for these services by law, implementation of these services is difficult, costly, and often fails to meet the goals of the Act. Despite shortcomings, the Act has been shown to help reduce recidivism and provide temporary employment (Bureau of Justice Assistance, U.S. Department of Justice, 2013).

However, having a reentry location within a county may be related to increased rates of STDs. For example, a county with a reentry location may have a higher rate of individuals with STDs because of the ex-offenders' exposure while incarcerated. When ex-offenders are sent to reentry locations, they are free to engage in sexual relationships with other members of the community. Yet, they often have limited access to health insurance, face disruptions in their romantic relations, and have higher likelihoods of engaging in high-risk sexual behaviors such as selling sex for drugs or money. Research bears this out. Using interview data collected from 106

men recruited from 5 different prisons, Morrow and The Project Start Study Group (2009:238) found that 37% of the men reported having an STD, 79% reporting have sex within a week of release, and 22% reported having multiple partners and limited use of condoms within weeks of release. Thus, it is reasonable to expect that: *Hypothesis 10*: Net of other factors, counties with reentry facilities will have higher rates of chlamydia and gonorrhea compared with counties without reentry facilities.

## **Data and Methods**

### **Data**

The analysis includes all U.S. counties that reported data on chlamydia infections, gonorrhea infections, and racial composition (N=3089). It relies on indicators from various sources measured at the county level, including the Centers for Disease Control (2009 STD Surveillance System and the Bridged-Race Population Estimates), the U.S. Census Bureau (Counties Data Files, 2005-2007 and 2006-2010 American Community Surveys, and the Small Area Income and Poverty Estimates Program), and the Health Indicators Warehouse. The dependent variables—chlamydia rates and gonorrhea rates—were the averages of reported cases from 2007, 2008, 2009, and 2010. All other variables in the analysis except for reentry locations were the most recent indicators from various sources available prior to 2007. Reentry locations were those reported in the Residential Reentry Centers Directory by the Federal Bureau of Prisons in 2013. These indicators have been compiled into a single dataset in which county is the unit of analysis. Indicators include the rate of gonorrhea infection per 100,000, the rate of chlamydia infection per 100,000 and residential segregation (i.e., the black isolation index and the white isolation index).

The analysis relies on county-level data for several reasons. As McLaughlin and Stokes (2002: 100) suggest, although:

neighborhoods may be important in metropolitan counties, nonmetropolitan residents are more likely to view the county as an important economic and social unit. The availability and accessibility of health care and of educational, civic, cultural, job, environmental, and recreational opportunities are largely determined at the local level and influenced by local structures. The county is often the decision-making unit for providing and organizing local services (McLaughlin and Stokes, 2002: 100).

Moreover, in order to understand how these dynamics operate in the national context, it is preferable to look beyond metropolitan areas only. Using county-level indicators provides the ability to carry out nationwide analysis. For analysis purposes, “all parishes, boroughs and census area county-equivalents are considered counties ... all independent cities have been consolidated into (joined to) the broader surrounding geographic county” (Rand, 2011: 6). Below, the operationalizations of the variables used in the analysis are included.

### **Operationalizations**

Chlamydia rate refers to the number of reported chlamydia cases in the county per 100,000 residents.

Gonorrhea rate refers to the number of reported gonorrhea cases in the county per 100,000 residents.

Lockup rate refers to the number of residents in correctional facilities within a county divided by the number of total residents in the county.



Reentry Location refers to whether a Bureau of Prisons ex-offender reentry facility (i.e., halfway houses for ex-offenders) is located in the county (coded 1 if yes and 0 otherwise).

This chapter uses the isolation index as an indicator of residential segregation because it is the most appropriate measure to use when studying infectious diseases (Acevedo-Garcia, 2000; and Pugsley, 2013). “Isolation measures the extent to which a member of a racial or ethnic group is likely to be in contact with members of the same group (as opposed to members of other groups)” (Acevedo-Garcia, 2000: 1154). Black isolation, therefore, is the extent to which blacks are likely to be in contact with other blacks as opposed to whites and other racial groups. Similarly, white isolation is the extent to which whites are likely to be in contact with other whites as opposed to other racial groups. Research has shown that high black isolation (i.e., greater than 60 on a scale from 0 to 100) is related to the concentration of disadvantages such as concentration of disease, crime, and unemployment (Massey and Denton, 1993; Bobo and Zubrinsky, 1996; Meyer, 2000; and Charles-Zubrinsky, 2003). White isolation, on the other hand, is associated with beneficial outcomes in health, wealth, and political power by isolating whites from communities with concentrated disadvantage (Beaulieu and Continelli, 2011).

In addition, counties were coded to indicate their racial composition. “Race of county” is a heuristic device used to categorize counties according to their percentage of black residents. Following the work of Benjamins et al. (2004), for each county, the percentage black residents was determined and coded to indicate whether that percentage was in the bottom quartile (i.e., less than .7% black residents), the middle two quartiles (i.e., between .7% and 10.3% black residents), or the top quartile (i.e., more than 10.3% black residents). For the convenience of the reader, these quartiles are referred to as communities with the lowest percentages of Black

residents, communities with intermediate percentages of Black residents, and in communities with the highest percentages of Black residents.

Percent Latino. For each county, the percentage of residents who are Latino or Hispanic.

Sex Ratio. For each county, the number of male residents per 100 female residents.

Unemployment Rate. People are classified as unemployed if they do not have a job, have actively looked for work in the prior four weeks, and are currently available for work. The percent unemployed is the number of 16 year-old and over residents in the county per 100 16 year-old and over residents in the county.

Percent Immigrant. For each county, the percentage of residents who were not born in the United States.

Median Income. For each county, the median income is the dollar amount that divides the income distribution into two equal groups such that half of the population has income above that amount and of the population has income below that amount.

Shortage of Health Professionals. A county is designated as having a shortage of health professionals when it is an urban or rural area that has “a population to full-time equivalent primary care physician ratio ... greater than 3,000:1 and have unusually high needs for primary care services or insufficient capacity of existing primary care providers ... [or that] demonstrate that primary medical professionals ... are overutilized, excessively distant, or inaccessible to the population under consideration.” (Taylor, 2004: 11). Counties that were designated as having a shortage of health professionals were coded 1, and others were coded 0.

Population Density. For each county, the number of residents per square mile.

Percent College Graduates. For each county, the percentage of residents 25 years old or older who have a college degree.

Income Inequality (i.e., Gini Coefficient). The Gini coefficient is a measure of statistical dispersion that measures the inequality among values of household income. It is computed in such a way that a value of 0 indicates perfect income equality such that all values are the same and everyone has an exactly equal income and a value of 100 indicates maximum inequality such that one family has all the income inequality.

### **Analysis Strategy**

Stata 12.0 was used to carry out the data analysis and data management. The analysis is based on a series of Ordinary Least Squares (OLS) regression models in which community rates of chlamydia and gonorrhea are the dependent variables. The central independent variables are lockup rate and reentry location. In addition, the models take into consideration black isolation, white isolation, college graduation rates, percent Latino residents, sex ratios, unemployment rates, median income, percent immigrant residents, population density, and income inequality (i.e., the Gini index) as predictors of chlamydia rates and gonorrhea rates. The analysis also examines how these factors are related to chlamydia rates and gonorrhea rates when stratified by racial composition of county. The stratified analyses provide some assessment of how great the disparities in STDs are between communities with the lowest percentages of black residents and communities with the highest percentages of black residents, net of other community health-related factors.

Several diagnostics were performed in order to test for multicollinearity, homoscedasticity, and model specification. To test for multicollinearity, I used two diagnostic tests. Using the Stata correlate command to examine Pearson product-moment correlation

coefficients for all pairs of variables used in the analyses (i.e., a correlation matrix). Generally, a correlation matrix indicates whether the independent variables to be used in the analyses are not too highly correlated so that they create problems of collinearity (Kleinbaum et al., 1987). Correlations greater than  $\pm .5$  may be problematic inasmuch as they may lead to unreliable estimates. When conducting multivariate analyses, I used the Stata variance inflation factor command (VIF) to scrutinize the correlations of the independent variables in greater detail. When values on this test exceeds 10.0, the correlations among the independent variables may be too great, and thus, indicate unreliable estimates (Institute for Digital Research and Education, 2012).

An assumption of OLS Regression is that of homoscedasticity—i.e., that the variance of errors is the same across all levels of the dependent variable. In order to test for heteroscedasticity, (i.e., the violation of the assumption of homoscedasticity), I use the Breusch-Pagan test for heteroscedasticity. This test assesses conditional heteroscedasticity, and it suggests that there may be problems with conditional heteroscedasticity when the chi-square value for the test has a probability value less than .05. When this occurs, either the model under consideration should be re-specified or it should be fit using robust standard errors (Berry and Feldman, 1985). Because all of the models run indicated the possibility of heteroscedasticity, the regressions were run using robust standard errors to correct for threats to stability and reliability (Berry and Feldman, 1985).

Model specification is assessed with a variety of test using the Stata command `fitstat`. Scott and Freese (2001:82) suggest that for all models, “`fitstat` reports the log-likelihoods of the full and intercept-only models, the deviance (D), the likelihood ratio chi-square (G2), Akaike’s

Information Criterion (AIC), AIC\*N, the Bayesian Information Criterion (BIC), and BIC`.”

They suggest that “fitstat is particularly useful for comparing two models.” They say that

“Akaike’s (1973) information criteria is defined as  $AIC = \frac{-2 \ln \hat{L}(M_k) + 2P}{N}$ , where  $\hat{L}(M_k)$  is the likelihood of the model and  $P$  is the number of parameters in the model .... All else being equal, the model with the smaller AIC is considered the better fitting model” (Scott and Freese, 2001: 86).

In contrast, Scott and Freese (2001: 86) suggest that the Bayesian information criterion or BIC is “a measure of overall fit and a means to compare nested and non-nested models. ... BIC is defined as  $BIC_k = D(M_k) - df_k \ln N$  where  $df_k$  is the degrees of freedom associated with the deviance. The more negative the  $BIC_k$ , the better the fit.”

Stata’s fitstat command also produces an adjusted coefficient of determination ( $R^2$ ) statistic, which offers an indication of model fit. According to Scott and Freese (2001:84) The  $R^2$  can be defined as:

$$R^2 = 1 - \frac{\sum_{i=1}^N (y_i - \hat{y}_i)^2}{\sum_{i=1}^N (y_i - \bar{y})^2} = \frac{\widehat{\text{Var}}(\hat{y})}{\widehat{\text{Var}}(\hat{y}) + \widehat{\text{Var}}(\hat{\varepsilon})} = 1 - \left[ \frac{L(M_{\text{Intercept}})}{L(M_{\text{Full}})} \right]^{2/N}$$

Similarly, the “adjusted  $R^2$  is defined as:

$$\overline{R}^2 = \left( R^2 - \frac{K}{N-1} \right) \left( \frac{N-1}{N-K-1} \right)$$

where  $K$  is the number of independent variables (Scott and Freese: 2001:84). This statistic provides a measure of how well observed outcomes are estimated by the model. It offers an indication of the proportion of total variation of outcomes explained by the model. Generally, the larger the adjusted  $R^2$ , the better the model fit.

As mentioned above, the analysis is based on a series of OLS regression models in which community rates of chlamydia and gonorrhea are the dependent variables. The following OLS regression models are estimated:

$$7. \text{ Chlamydia Rate} = \alpha + \beta_1 * \text{Lockup Rate} + \beta_2 * \text{Reentry Location} + \beta_3 * \text{Black Isolation} + \beta_4 * \text{White Isolation} + \beta_5 * \text{Percent College Graduates} + \beta_6 * \text{Percent Latino} + \beta_7 * \text{Sex Ratio} + \beta_8 * \text{Percent Unemployed} + \beta_9 * \text{Median Income} + \beta_{10} * \text{Percent Immigrant} + \beta_{11} * \text{Health Professional Shortage} + \beta_{12} * \text{Population Density} + \beta_{13} * \text{Gini Index} + \varepsilon$$

This equation suggests that chlamydia rates are a function of lockup rates, reentry locations, black isolation, white isolation, percent college graduates, percent Latino residents, sex ratios, percent unemployed, median income, percent immigrant residents, whether there is a health professional shortage, population density, and income inequality.

$$8. \text{ Chlamydia Rate} = \alpha + \beta_1 * \text{Lockup Rate} + \beta_2 * \text{Reentry Location} + \beta_3 * \text{Black Isolation} + \beta_4 * \text{White Isolation} + \beta_5 * \text{Percent College Graduates} + \beta_6 * \text{Percent Latino} + \beta_7 * \text{Sex Ratio} + \beta_8 * \text{Percent Unemployed} + \beta_9 * \text{Median Income} + \beta_{10} * \text{Percent Immigrant} + \beta_{11} * \text{Health Professional Shortage} + \beta_{12} * \text{Population Density} + \beta_{13} * \text{Gini Index} + \varepsilon \mid \text{Percent Black Residents}$$

This equation suggests that chlamydia rates in communities with various percentages of Black residents are a function of black isolation, white isolation, percent college graduates, percent Latino residents, sex ratios, percent unemployed, median income, percent immigrant residents, whether there is a health professional shortage, population density, and income inequality.

$$9. \text{ Gonorrhea Rate} = \alpha + \beta_1 * \text{Lockup Rate} + \beta_2 * \text{Reentry Location} + \beta_3 * \text{Black Isolation} + \beta_4 * \text{White Isolation} + \beta_5 * \text{Percent College Graduates} + \beta_6 * \text{Percent Latino} + \beta_7 * \text{Sex Ratio} + \beta_8 * \text{Percent Unemployed} + \beta_9 * \text{Median Income} + \beta_{10} * \text{Percent Immigrant} + \beta_{11} * \text{Health Professional Shortage} + \beta_{12} * \text{Population Density} + \beta_{13} * \text{Gini Index} + \varepsilon$$

This equation suggests that Gonorrhea rates are a function of lockup rates, reentry locations, black isolation, white isolation, percent college graduates, percent Latino residents, sex ratios, percent unemployed, median income, percent immigrant residents, whether there is a health professional shortage, population density, and income inequality.

$$10. \text{Gonorrhea Rate} = \alpha + \beta_1 * \text{Lockup Rate} + \beta_2 * \text{Reentry Location} + \beta_3 * \text{Black Isolation} + \beta_4 * \text{White Isolation} + \beta_5 * \text{Percent College Graduates} + \beta_6 * \text{Percent Latino} + \beta_7 * \text{Sex Ratio} + \beta_8 * \text{Percent Unemployed} + \beta_9 * \text{Median Income} + \beta_{10} * \text{Percent Immigrant} + \beta_{11} * \text{Health Professional Shortage} + \beta_{12} * \text{Population Density} + \beta_{13} * \text{Gini Index} + \varepsilon | \text{Percent Black Residents}$$

This equation suggests that gonorrhea rates in communities with various percentages of Black residents are a function of lockup rates, reentry locations, black isolation, white isolation, percent college graduates, percent Latino residents, sex ratios, percent unemployed, median income, percent immigrant residents, whether there is a health professional shortage, population density, and income inequality.

### **Preliminary Diagnostics and Preliminary Results**

As an initial check for problems of multicollinearity, the analysis examined the correlations among the variables used in the analysis. The correlation matrix shows the Pearson product-moment correlation coefficients for all pairs of variables used in the analyses (see Appendix F). It shows that there are 3,089 observations when using listwise deletion (i.e., the entire observation is omitted from the estimation sample if any of the variables in to be used in the analyses is missing for that observation) (Bruin, 2006). The correlation matrix shows that all but two correlations that are less than .5. Generally, this suggests that the independent variables to be used in the analyses are not too highly correlated so that they create problems of collinearity (Kleinbaum et al., 1987). The correlations between lockup rate and sex ratio ( $r=$

.676), the gini index and black isolation ( $r = .508$ ), and between percent Latino and white isolation ( $r = -.786$ ) are potentially problematic. These correlations, along with others, will be scrutinized in greater detail with variance inflation factor (VIF) analysis in the multivariate analysis.

Table 4.1 presents the rates of chlamydia and gonorrhea per 100,000 residents by whether the county contains a reentry location. It shows that the overall rates of chlamydia are 407.8 per 100,000 residents. It also shows that in counties with reentry locations the rate is 544.3 per 100,000. This contrasts to 328.3 per 100,000 in counties without reentry locations. The gap in chlamydia rates between counties with reentry locations and those without such reentry locations is 216 cases per 100,000. Similarly, the chart shows that, for all counties, the gonorrhea rate is 99.0 per 100,000. In counties with reentry locations, the rate is 146.0 per 100,000. This contrasts to 71.6 per 100,000 in counties without reentry locations. The gap in gonorrhea rates between counties with reentry locations and those without such reentry locations is 74 cases per 100,000.

Table 4.1 presents selected characteristics of counties. In the final column (“All Counties”), it presents selected characteristics for all counties. It again shows that, for all counties, the chlamydia rate is 407.8 per 100,000, and the overall gonorrhea rate is 99.0. It also shows that the average black isolation index score is 26.7, and the average white isolation index score is 77.4. Overall, 14.4% of counties are located in the Northeast, 26.1% are in the Midwest, 36.7% are in the South, and 22.9% are located in the West. The average percentage of college graduates is 28.1. The average percentage of Latinos is 15.7. On average, there are 96.7 men per 100 women. The average unemployment rate is 8.0 percent. The overall median income is \$52,313. The percentage immigrant is 7.7. It shows that 4.5 percent of counties have a health



care professional shortage. The average population density is 223.3 people per square mile. The average Gini index score is 44.4.

A central concern of this analysis is whether counties with reentry locations differ from those without such reentry locations in their rates of chlamydia and gonorrhea. It is, therefore, appropriate to determine whether these counties differ on characteristics that might be related to chlamydia rates and gonorrhea rates.

Table 4.1 shows that counties differ by selected characteristics. For example, counties with reentry locations have significantly higher black isolation scores (38.9) than do counties without such reentry locations (19.5). In contrast, counties with reentry locations have significantly lower white isolation index scores (69.8) than do those without reentry locations (81.9).

Counties with reentry locations are disproportionately located in the West. Counties with reentry locations are underrepresented among those with health care professional shortages. On average, counties with reentry locations have higher percentages of college graduates, higher percentages of Latino residents, greater male-to-female sex ratios, lower median incomes, higher percentages of immigrant residents, more dense populations, and greater income inequality than do those without such reentry locations. These factors are statistically controlled in the analysis.

## **Results**

This analysis will look at whether incarceration is related to county rates of chlamydia and gonorrhea. How are lockup rates and reentry locations related to rates of chlamydia and gonorrhea? Do these relationships vary by racial composition of county? The results of the analysis are presented below.

Table 4.2 presents ordinary least squares (OLS) regression models predicting chlamydia rates with lockup rates and reentry location. Model 1 shows that as the lockup rate increases by one, chlamydia rates increase by 6.7 per 100,000 ( $p < .001$ ). In addition, on average counties with reentry locations have chlamydia rates that are 218.1 cases per 100,000 higher than counties without reentry locations. These patterns are fully consistent with Hypotheses 9 and 10. Lockup rates and reentry locations account for more than 18% of the variance in chlamydia rates. The AIC of 13.6 (versus 13.8 for the null model) and the BIC of 17509.8 (versus 18135.7 for the null model) suggest that the model provides a better fit of the data. Moreover, the VIF of 1.0 suggests that the independent variables are not too highly correlated to raise issues of reliability.

What happens to these patterns when other factors are taken into account? Model 2 of Table 4.2 presents OLS regression models predicting chlamydia rates with lockup rates and reentry locations, net of other county factors. Model 2 shows that, net of reentry locations, black isolation, white isolation, percent college graduates, percent Latino, sex ratio, percent unemployed, median income, percent immigrant, healthcare professional shortage designation, population density, and income inequality, lockup rates are not systematically related to chlamydia rates ( $p > .5$ ). On average, counties with reentry locations have chlamydia rates that are 68.7 cases higher than comparable counties without reentry locations ( $p < .001$ ). Model 2 also shows that other factors are related to chlamydia rates. In particular, it shows that as black isolation increases by one unit, chlamydia rates increase by 4.4 cases per 100,000 ( $p < .001$ ). In contrast, as white isolation increases by one unit, chlamydia rates decrease by 7.6 per 100,000 ( $p < .001$ ). Like much previous literature, these results suggest that black isolation, as an indicator of concentrated disadvantage, is associated with increasing rates of STDs. However, the results

also show that white isolation operates in a distinctly different fashion such that it is associated with decreasing rates of chlamydia.

Model 2 also shows that other factors are related to chlamydia rates. In particular, it shows that net chlamydia rates are significantly higher in the West than in the South. It also shows that chlamydia rates increase when the percent Latino residents decreases, the unemployment rate increases, median income decreases, population density increases, and income inequality increases. Net of other factors, counties with health professional shortages have lower chlamydia rates. Combined, these factors account for 65.5% of the variance in chlamydia rates in counties. The AIC and BIC show improvements over the incarceration-only model, and the VIF shows that the independent variables are not too highly correlated.

Recall that Chapter 3 showed that the relationship between racial isolation and chlamydia rates varies by the racial composition of the county. Therefore, this analysis also presents the results stratified by racial composition of county. Model 1 of Table 4.3 shows that, net of other county-level factors, in counties with the lowest percentages of Black residents, lockup rates and reentry locations are not systematically related to chlamydia rates. Black isolation is not systematically related to chlamydia rates, but white isolation is associated with decreases in chlamydia rates, net of other factors such as region, education, percent Latino, sex ratios, percent unemployed, median income, percent immigrant, health professional shortage, population density, and income inequality. These results suggest that, in the context of counties with the lowest percentages of Black residents, several factors usually associated with concentrated disadvantage (e.g., lower education, lower income, and greater income inequality) appear not to be related to chlamydia rates in counties with the lowest percentages of Black residents as they

are in the general population. In counties with the lowest percentages of Black residents, there is no systematic relationship between education and chlamydia rates, sex ratios and chlamydia rates, income and chlamydia rates, nor income inequality and chlamydia rates. These factors account for 42.6% of the variance in chlamydia rates in counties with the lowest percentages of Black residents. It should be noted that because the VIF shows that percent Latino and white isolation were too highly correlated, percent Latino was omitted from the model.

Model 2 shows that, in counties with intermediate percentages of Black residents, net of other county factors, higher lockup rates are associated with lower rates of chlamydia ( $p < .001$ ). This finding is not consistent with Hypothesis 9. However, counties with reentry locations have significantly higher rates of chlamydia ( $p < .001$ ). This finding is fully consistent with Hypothesis 10. Black isolation is associated with higher rates of chlamydia, but white isolation is associated with lower rates of chlamydia. In this context, the relationships between chlamydia rates and region, unemployment, median income, percentage immigrant residents, health professional shortages, and population density are also similar to those in the general analysis.

Model 3 shows that lockup rates are not systematically related to chlamydia rates in disproportionately black counties. Still, disproportionately black counties with reentry locations have significantly higher chlamydia rates. Black isolation is associated with higher rates of chlamydia, but white isolation still associated with lower rates of chlamydia in counties with the highest percentages of Black residents. Again, factors such as percentage Latino residents, sex ratio, unemployment rate, median income, and income inequality are related to chlamydia rates in ways that are similar to the patterns for the general analysis. These results in Models 2 and 3 are not consistent with Hypothesis 9 but fully consistent with Hypothesis 10. In both Models 2

and 3, these factors account for more than 56% of the variance in chlamydia rates in counties with intermediary and the highest percentages of Black residents.

Table 4.4 presents ordinary least squares (OLS) regression models predicting gonorrhea rates with lockup rates and reentry location. Model 1 shows that as the lockup rate increases by one, gonorrhea rates increase by 2.0 per 100,000 ( $p < .05$ ). In addition, on average counties with reentry locations have gonorrhea rates that are 75.0 cases per 100,000 higher than counties without reentry locations. These patterns are fully consistent with Hypotheses 9 and 10. Lockup rates and reentry locations account for more than 13% of the variance in gonorrhea rates. The AIC of 11.86 (versus 12.0 for the null model) and the BIC of 11984.6 (versus 12424.2 for the null model) suggest that the model provides a better fit of the data. Moreover, the VIF of 1.0 suggests that the independent variables are not too highly correlated to raise issues of reliability.

Model 2 of Table 4.4 shows that when other factors are taken into account, the higher the lockup rates are, the lower the gonorrhea rates are ( $p < .05$ ). This finding is not consistent with Hypothesis 9. Model 2 also shows that, on average, counties with reentry locations have gonorrhea rates that are 21.1 cases higher than comparable counties without reentry locations ( $p < .01$ ). Model 2 also shows that other factors are related to gonorrhea rates. In particular, it shows that as black isolation increases by one unit, gonorrhea rates increase by 2.35 cases per 100,000 ( $p < .001$ ). In contrast, as white isolation increases by one unit, gonorrhea rates decrease by 1.97 per 100,000 ( $p < .001$ ). In other words, while these results suggest that black isolation, as an indicator of concentrated disadvantage, is associated with increasing rates of STDs, white isolation operates in a distinctly different fashion such that it is associated with decreasing rates of gonorrhea.

Model 2 also shows that net of other factors, gonorrhea rates are significantly lower in the Northeast but higher in the Midwest than in comparable counties in the West. The model also shows that gonorrhea rates increase when the percent Latino residents decreases, the unemployment rate increases, median income decreases, and income inequality increases. Net of other factors, counties with health professional shortages have lower gonorrhea rates. Combined, these factors account for more than 70% of the variance in gonorrhea rates in counties. The AIC and BIC show improvements over the racial isolation-only model, and the VIF shows that the independent variables are not too highly correlated.

Table 4.5 presents the analysis stratified by racial composition of county. Model 1 of Table 4.5 shows that, net of other factors, in counties with the lowest percentages of Black residents, lockup rates are not systematically related to gonorrhea rates ( $p > .5$ ). This model also suggests that in counties with the lowest percentage black residents, the presence of reentry locations is not systematically related to gonorrhea rates ( $p > .4$ ).

Model 1 also shows that neither black isolation nor white isolation is systematically associated with gonorrhea rates, net of other factors such as region, education, sex ratios, percent unemployed, median income, percent immigrant, health professional shortage, population density, and income inequality. These factors account for 18% of the variance in gonorrhea rates in counties with the lowest percentages of Black residents. The AIC and BIC show improvements over the incarceration-only model. Again, because the VIF shows that percent Latino and white isolation were too highly correlated, percent Latino was omitted from the model.

Model 2 of Table 4.5 shows that, in counties with intermediate percentages of Black residents, net of other county factors, higher lockup rates are associated with lower gonorrhea rates ( $p < .001$ ). Counties with reentry locations have higher gonorrhea rates ( $p < .001$ ). These results are not consistent with Hypotheses 9, but they provide support for Hypothesis 10. Also, black isolation is associated with higher rates of gonorrhea, but white isolation is associated with lower rates of gonorrhea. In this context, the relationships between gonorrhea rates and unemployment, median income, percentage immigrant residents, health professional shortages, and population density are also similar to those in the general analysis. These factors account for 54.7% of the variance in gonorrhea rates in counties with intermediary percentages of Black residents. The AIC and BIC show improvements over the incarceration-only models, and the VIF shows that the independent variables are not too highly correlated.

Model 3 of Table 4.5 shows that in counties with the highest percentages of Black residents, neither lockup rates nor reentry locations are systematically related to gonorrhea rates. These patterns are not supportive of Hypotheses 9 nor 10. Net of other factors, black isolation is associated with higher rates of gonorrhea, but white isolation is associated with lower rates of gonorrhea. Again, factors such as percentage Latino residents, sex ratio, unemployment rate, median income, and income inequality are related to gonorrhea rates in ways that are similar to the patterns for the general analysis. Combined these factors account for 61% of the variance in gonorrhea rates in counties with the highest percentages of Black residents. In both Models 2 and 3, the AIC and BIC show improvements over the incarceration-only models, and the VIF shows that the independent variables are not too highly correlated.

Generally, there is little support for Hypothesis 9 that higher lockup rates are associated with higher STD rates. Lockup rates are not systematically related to chlamydia rates; moreover, they are negatively related to gonorrhea rates. There is substantially more support for Hypothesis 10 that counties with reentry locations will have higher rates of chlamydia and gonorrhea. Counties with reentry locations do have significantly higher rates of chlamydia and gonorrhea. The stratified analysis showed that counties with reentry locations have higher chlamydia and gonorrhea rates except in counties with the lowest percentages of Black residents.

The results of the analysis of gonorrhea rates are slightly different from those for chlamydia rates. In particular, lockup rates are not systematically related to chlamydia rates; however, they are negatively related to gonorrhea rates. Also, in counties with the highest percentages of Black residents, counties with reentry locations have higher chlamydia rates, but they do not have higher gonorrhea rates. In other words, the results are less supportive of Hypotheses 9 and 10 with respect to gonorrhea than with chlamydia.

Chapter 3 estimated overall chlamydia rates and gonorrhea rates, chlamydia and gonorrhea rates by racial composition of county, and chlamydia and gonorrhea rates by racial composition of county if black isolation could be eliminated. Figure 4.1 presents predicted chlamydia rates by racial composition of county, with and without reentry locations, and if black isolation equaled 0 in counties without reentry locations. This chart shows that the overall rates of chlamydia would decline by more than 35% (down from 407.8 to 257.9) if Black isolation were to be reduced to zero in counties without reentry locations. It also shows that rates in counties with the highest percentages of Black residents would decline by more than 180 cases (down from 560.1 to 376.5). The chlamydia rates in counties with the lowest percentages of



Black residents would decrease by 2 cases (down from 227.9 to 225.6). Also, chlamydia rates in counties with intermediate percentages of Black residents would fall by 32 cases (down from 251.9 to 219.6). In this scenario, the gap between white counties and disproportionately black counties would be reduced from 332 cases down to 151 cases.

Figure 4.2 presents predicted gonorrhea rates by racial composition of county, with and without reentry locations, and if black isolation equaled 0 in counties without reentry locations. This chart shows that overall gonorrhea rates would decline by more than 63 cases (down from 99.0 to 35.8). It also shows that rates in counties with the highest percentages of Black residents would decrease by more than 98 (down from 171.0 to 72.2). The gonorrhea rates in counties with the lowest percentages of Black residents would increase by 9.8 (up from 14.6 to 24.4). However, the gonorrhea rates in counties with intermediate percentages of Black residents would fall by more than 24 cases (down from 48.4 to 23.9). If black isolation could be eliminated and reentry locations moved out of disproportionately black counties, it is estimated that the gap in gonorrhea rates between white counties and disproportionately black counties could be reduced from 84 cases to 48 cases per 100,000 residents.

These results illustrate the combined power of residential segregation and reentry locations in perpetuating disparities in chlamydia rates and gonorrhea rates between counties with the lowest percentages of Black residents and counties with the highest percentages of Black residents.

### **Discussion and Conclusions**

This chapter began with a focus on the connection between policies such as the “War on Drugs,” mass incarceration, prisoner reentry locations, and STD disparities. With millions of

formerly incarcerated individuals disproportionately concentrated in disadvantaged communities, this chapter examined whether incarceration and reentry matter differently for different types of counties. It also explores whether predicted rates of STDs would decrease if reentry and residential segregation were eliminated.

The chapter pointed out that, compared with those in the general public, those in prison have rates of infectious diseases such as gonorrhea and chlamydia that are 4 to 10 times higher. Sexual activity among those incarcerated is a relatively common practice. The federal government has reported that at least 30% of those incarcerated engage in some form of sexual behavior (Federal Bureau of Prisons, 2010). There are also other forms of transmission of STDs beyond simple sexual intercourse such as bites, cuts, and physical attacks. When prisoners face such risks, sexual behavior modifications do little to reduce the spread of STDs.

Given the lack of treatment and high rates of STDs in prisons, the communities to which prisoners reenter are also at elevated risk. More than 700,000 inmates are released each year from prisons and back into communities (Massoglia et al., 2012). Despite these record numbers, few services are in place to address their material, emotional, or health needs. Without proper access to sexual healthcare while incarcerated or once they return to their communities, both individuals and communities are placed at an increased risk of STDs. When those who were formerly incarcerated return to their communities they are often at a disadvantage to meet the most basic of human needs. Such deprivation often leads these individuals to engage in high risk sexual behaviors that further exacerbate the STD rate within their communities (Schnittker and John, 2007).

Using county-level data, this chapter tested two hypotheses. Hypothesis 9 stated that as incarceration rates in counties increase, the rates of chlamydia and gonorrhea will increase. Generally, there is little support for Hypothesis 9 that higher lockup rates are associated with higher STD rates. Although bivariate analysis provided support, multivariate analysis led to the rejection of this hypothesis, as lockup rates are not systematically related to chlamydia rates; moreover, they are negatively related to gonorrhea rates. The lack of support for this hypothesis is likely due to the fact that the indicator of incarceration—lockup rate—measures incarceration rates for the counties in which inmates are detained, rather than the counties of origin of inmates. This is consequential because “while most prisoners in America are from urban communities, most prisons are now in rural areas” (Huling, 2002: 197). As Huling (2002: 197) suggests, “huge numbers of inmates from urban areas become rural residents for the purposes of Census-based formulas used to allocate government dollars and political representation,” but they are not systematically included in the enumeration of these locales’ STD rates. Thus, the presence of prisons in counties potentially inflates the population bases of these counties without elevating their rates of STDs. As a consequence, reentry locations may serve as a more relevant indicator of incarceration.

This chapter also provided an assessment of Hypothesis 10 that counties with reentry facilities will have higher rates of chlamydia and gonorrhea compared with counties without reentry facilities. The results showed that counties that contain ex-offender reentry locations have higher rates of STDs. As the chapter pointed out, reentry locations may have higher rates of STDs because relatively high proportions of their residents have been exposed while incarcerated. When ex-offenders are sent to reentry locations, they are free to engage in sexual

relationships with other members of the community. Yet, they often have limited access to health insurance, face disruptions in their romantic relations, and have higher likelihoods of engaging in high-risk sexual behaviors such as selling sex for drugs or money.

Finally, the chapter suggests that the presence of a reentry location is related to counties' STD rates. The results from this analysis suggest that relegating the formerly incarcerated to communities that already face concentrated disadvantage further exacerbates racial disparities in STDs. This research incorporates contextual variables that are consistent principles central to an intersectional approach that points to the structural solutions that are needed to reduce the rates of STDs generally and racial disparities in particular. Placing the blame on individuals has done little to solve the problem. Furthermore, concentrating disadvantage by maintaining white privilege through institutionalized segregation practices is one of the most important mechanisms by which racial disparities in community rates of STDs is sustained.

## **CHAPTER 5**

### **Review of Findings, Limitations, Conclusions, and Implications of Research**

#### **Review of the Problem**

This study began with the observation that there are approximately 19 million new cases of sexually transmitted diseases in the United States every year (CDC, 2012). It pointed out that STDs impose physical, psychological, and economic costs on individuals and society as a whole, but that the burden of these diseases is not equally endured along gender lines and racial lines. Generally, women have rates of chlamydia that are more than two and a half times the rate of men. Black women have the highest rates of chlamydia that are more than seven times the rate among white women. The chlamydia rate among black men was almost 11 times the rate among white men (CDC, 2010).

Despite the documented health disparities between Whites and African Americans, researchers have not fully unraveled the causes of such differences. Popular notions of STD disparities suggest that African American men and women engage in individual non-normative behaviors that explain their higher rates. However, research has shown that racial and gender disparities persist even when African Americans engage in similar sexual behaviors as Whites. The puzzle is this: A key to reducing sexually transmitted diseases is engaging in safe sex practices like condom use. But even though African Americans report higher rates of STDs, they report higher rates of condom use than Whites. African Americans also report lower rates of anal intercourse and somewhat lower rates of male same-sex intercourse. Thus, the question is: “If it is not just individual behaviors that explain disparities in sexually transmitted diseases, then what

is driving such disparities?” The failure of behavioral factors alone to explain STD disparities calls for further investigation.

This dissertation research has attempted to help solve the puzzle. The first part of my analysis used nationally representative data from the National Survey of Family Growth to carry out an individual-level analysis that tests whether factors based on sexual network theories help explain racial and gender disparities in the reporting of chlamydia, gonorrhea, or syphilis. The second part of the research was motivated by Massey and Denton’s (1993) *American Apartheid* hypersegregation theory. It used county-level data to look at the relationship between residential segregation and chlamydia rates. The third part of the analysis included consideration of incarceration and ex-offender reentry locations and their relationship to community rates of chlamydia and gonorrhea.

My work used an intersectional framework that intended to address the fact that the experiences and struggles of women of color have fallen between the cracks of both feminist and anti-racist discourse. It asserted that analyses need to examine complex social locations take both gender and race on and show how they interact to shape outcomes. The analysis used the complex social location of race by gender. My research sought to answer several questions concerning the relationship between sexual network factors and the likelihood of reporting an STD. It also examined the relationship between residential segregation and county rates of STDs, as well as the relationship between incarceration and ex-offender reentry locations and county rates of STDs. I put forth several questions: (1) Above and beyond individual and behavioral determinants of STDs, what sexual network factors are specifically related to racial disparities in STDs? (2) Do sexual network factors account for disparities in STDs? (3) How is residential

segregation, especially racial isolation, related to racial disparities in STDs? And (4) how are incarceration rates and ex-offender reentry locations related to STDs?

### **Summary of Findings**

In the first part of my analysis, I was particularly concerned with whether the gaps between African American women and other race by gender groups are explained when taking sexual network factors into account. Proponents of sexual network theory argue that the sexual networks in which individuals have sex influence their likelihood of contracting an STD above and beyond their personal behaviors, so they focus on the characteristics of people's sexual partners. For example, these theories suggest that having sex partners who have additional sex partners increases the likelihood of contracting an STD. Laumann and Youm (1999) argue that people engaging in what they think are low-risk behaviors may actually be at a higher risk for infection than those who are in casual sexual relationships if their partners are in concurrent relationships. Adimora and Schoenbach (2010) argue that African American women may be more tolerant of their partners being in concurrent relationships because of sex ratio imbalances and power imbalances. These imbalances may encourage acceptance of relationships with those who have concurrent relationships, and concurrent relationships place the various partners at greater risk. Thus, *Hypothesis 1* is that: Net of other factors, those who have partners in concurrent relationships are more likely to report an STD. *Hypothesis 1B*: Compared with African American women with concurrent partners, African American men are no more likely, but White women, White men, Latino women, Latino men, other race women and other race

men with concurrent partners are less likely to report an STD, net of individual, behavioral, and other sexual network factors.

Similarly, sexual network theory suggests that even if people do not personally engage in risky behaviors themselves, they can become exposed to greater risks for STDs if others in their sexual networks engage in risky behaviors such as participating in commercial sex, using illicit drugs, or having sex with people who are known to have an infection. If African American women are more likely to have sex with partners who engage in high-risk behaviors than are Whites, this should help account for racial disparities in STDs. Thus, *Hypothesis 2A* is that: Those who engage in sex with high-risk partners are more likely to report an STD. *Hypothesis 2B*: African American women with high risk partners are no more likely than are African American men, but more likely to report an STD compared with White women, White men, Latino women, Latino men, other race women and other race men with high risk partners, net of individual, behavioral, and other sexual network factors.

De et al. (2004) also suggest people who have multiple partners are at greater risk for infection. Core Members are respondents who have had four or more sexual partners in the past 12 months. Periphery Members are respondents who had 1 or no sexual partners in the past 12 months. Thus, *Hypothesis 3A*: Net of individual, behavioral, and other sexual network factors, core members are more likely to report an STD. *Hypothesis 3B*: African American women core members are no more likely than are African American men, but are more likely to report an STD compared with White women, White men, Latino women, Latino men, other race women, and other race men who are core members net of individual, behavioral, and other sexual network factors. *Hypothesis 4A*: Net of individual, behavioral, and other sexual network factors,



periphery members are likely to report an STD. *Hypothesis 4B*: African American women periphery members are no more likely than are African American men, but are more likely to report an STD compared with White women, White men, Latino women, Latino men, other race women, and other race men who are periphery members, net of individual, behavioral, and other sexual network factors.

Laumann and Youm (1999) suggest that “bridging” exists when members of one group such as African Americans have sex with members of another group, become infected, and then infect members of their own group. *Hypothesis 5A*: Net of individual, behavioral, and other sexual network factors, individuals who engage in racial bridging are no more likely to report an STD compared with those who do not engage in racial bridging. *Hypothesis 5B*: African American women who engage in racial bridging are no more likely than are African American men, but are less likely to report an STD, compared with White women, White men, Latino women, Latino men, other race women, and other race men who engage in racial bridging, net of individual, behavioral, and other sexual network factors.

Sexual network theories imply that if African American women were in networks that were similar to other social locations, after accounting for these sexual network factors, disparities between African American women and other race by gender groups should disappear. The results from multivariate logistic regression analyses suggest that, net of sociodemographic and behavioral factors, the main effects of the sexual network factors are consistent with the predictions derived from sexual network theory (i.e., four out of five hypotheses). However, factors associated with sexual network theory do not appear to go very far in terms of explaining the racial and gender *gaps* in STDs, as none of the sexual network by social location interactions

were consistent with hypotheses derived from sexual network theory. Sexual network by social location interactions do little to explain racial and gender disparities in STDs.

Despite the relevance of sexual networks in explaining differences in STDs, they do not help explain much about the racial and gender gaps in STDs. In part, this is because African American women, on average, are already *less* likely than many other social locations to have sex with partners who are involved in risky sexual practices, and they are also less likely than many other social locations to be core members of sexual networks. Moreover, such theories do not usually provide a compelling explanation of why it is that particular kinds of people end up in the networks in which they are embedded. Although they assert that sexual segregation matters in maintaining racial disparities in STDs, they do not give sufficient attention to socioenvironmental factors (e.g., residential segregation) which may also be related to sexual segregation and related to racial disparities in STDs.

The second part of the analysis focused on the relationship between racial segregation and community (county) rates of chlamydia and gonorrhea. Massey and Denton (1993) suggest that African Americans continue to suffer high rates of poverty and other deleterious outcomes because of systemic residential segregation. Hyper-segregation acts as a stratifying agent. They suggest that high levels of black isolation interact with other community-level characteristics such as low SES and sex ratio imbalances to concentrate disadvantage. Biello et al. (2007) and Pugsely et al. (2013) suggest that Black isolation is related to higher rates of STDs. However, they did not examine the role of white isolation, nor whether racial isolation is related to STDs differently in different types of communities.

My analysis examined the impacts of both black isolation *and* white isolation in virtually all-white counties, in integrated counties, and in disproportionately black counties. My analysis sought to determine whether white isolation has the opposite relationship to STDs by acting as a structural factor that is associated with advantages for those living in white-isolated areas. Based on the *American Apartheid* Hyper-segregation theory, I put forth *Hypothesis 6*: Net of other factors, black isolation is associated with increases in chlamydia rates, but white isolation is associated with lower chlamydia rates. *Hypothesis 7*: In white counties, both black isolation and white isolation are associated with decreases in chlamydia rates. *Hypothesis 8*: In integrated and in disproportionately Black counties, black isolation is associated with higher rates of chlamydia, but white isolation is associated with lower rates of chlamydia.

These results suggested that white isolation is related to lower chlamydia rates, irrespective of the racial composition of the county. This is a finding that is new to the STD literature. What is also interesting and a new finding is that in white counties, black isolation is also associated with lower chlamydia rates.

Another way of understanding these results is in terms of disparities in STD rates by racial composition of county. The chlamydia rate for disproportionately Black counties is 563 cases per 100,000 residents. The chlamydia rate for white counties is 205. This suggests a disparity of 358 cases per 100,000 residents. If black isolation were reduced to 0, it is estimated that the overall rates of chlamydia would decline by more than 33%, down from 407.8 to 272.5. Chlamydia rates for disproportionately Black counties would decline by more than 25%, down from 563 to 411. This would shrink the disparity in rates between disproportionately Black counties and white counties by more than 150 cases without substantially increasing the rates in

white counties. The chlamydia rates in white counties would increase from 205 to 211. However, chlamydia rates in integrated counties would fall by more than 20%, down from 300 to 235.

In terms of gonorrhea, the rate for communities with the highest percentages of Black residents was 171.0 and the rate for communities with the lowest percentages of Black residents was 14.6. This suggests a disparity of 156.4 cases or a rate that is more than 11.7 times higher in communities with the highest percentages of Black residents. If black isolation were reduced to 0, it is estimated that the gonorrhea rates for communities with the highest percentages of Black residents would be 76.2 and the rates for communities with the lowest percentages of Black residents would be 21.8. This would yield a disparity of 54.4 cases or a rate that is 3.5 times higher in communities with the highest percentages of Black residents. In other words, the black county-white county chlamydia and gonorrhea rates disparities would be greatly reduced if black isolation were eliminated.

Overall, these results illustrate how powerfully residential segregation is related to STD rates. They show the role of black isolation in perpetuating disparities in chlamydia rates and gonorrhea rates between counties with the lowest percentages of Black residents and counties with the highest percentages of Black residents.

The third part of the analysis included consideration of incarceration and ex-offender reentry locations and their relationship to county rates of chlamydia and gonorrhea. According to Thomas et al. (2010), the inequitable removal of young African American men from their community through mass imprisonment shifts resources to white rural counties. This happens because federal policies allow counties where prisoners are housed to collect the political and economic resources associated with increased population due to the presence of those detained in

prisons. This reduces resources in the home communities of those incarcerated that could be used to prevent the spread of STDs through screening and testing.

Mass incarceration also has consequence of shifting sex ratios and affecting sexual network patterns both when men and women are incarcerated and reenter their communities (Thomas et al, 2010). Thus, mass incarceration also shifts sexual/dating patterns within communities. This suggests that incarceration plays a role in racial disparities above and beyond the individual level. *Hypothesis 9*: The higher the incarceration rates in counties are, the higher are the rates of chlamydia and gonorrhea.

In addition, more than 2 million people are released each year from jails and prisons to American communities. Because “more than 95% of incarcerated individuals eventually reenter the general community, amplification of infectious diseases during incarceration poses definite risks to the communities to which infected and untreated inmates return” (Awofeso, 2010: 27). Accordingly, I tested the proposition that having a reentry location within a county may be related to increased rates of STDs. *Hypothesis 10*: Net of other factors, counties with reentry facilities will have higher rates of chlamydia and gonorrhea compared with counties without reentry facilities.

Generally, there is little support for Hypothesis 9 that higher lockup rates are associated with higher STD rates. Lockup rates are not systematically related to chlamydia rates; moreover, they are negatively related to gonorrhea rates. There is substantially more support for Hypothesis 10 that counties with reentry locations will have higher rates of chlamydia and gonorrhea. A stratified analysis showed that counties with reentry locations have higher chlamydia and gonorrhea rates except in counties with the lowest percentages of Black residents.

The results also suggest that the overall rates of chlamydia would decline by more than 35% (down from 407.8 to 257.9) if Black isolation were to be reduced to zero in counties without reentry locations. It also shows that rates in communities with the highest percentages of Black residents would decline by more than 180 cases (down from 560.1 to 376.5). The chlamydia rates in communities with the lowest percentages of Black residents would decrease by 2 cases (down from 227.9 to 225.6). Also, chlamydia rates in communities with intermediate percentages of Black residents would fall by 32 cases (down from 251.9 to 219.6). In this scenario, the gap between white counties and disproportionately black counties would be reduced from 332 cases down to 151 cases.

The results also suggest that the overall rates of chlamydia would decline by more than 35% (down from 407.8 to 257.9) if Black isolation were to be reduced to zero in counties without reentry locations. It also shows that rates in communities with the highest percentages of Black residents would decline by more than 180 cases (down from 560.1 to 376.5). The chlamydia rates in communities with the lowest percentages of Black residents would decrease by 2 cases (down from 227.9 to 225.6). Also, chlamydia rates in communities with intermediate percentages of Black residents would fall by 32 cases (down from 251.9 to 219.6). In this scenario, the gap between white counties and disproportionately black counties would be reduced from 332 cases down to 151 cases.

The results suggested a similar decline in gonorrhea rates. If black isolation equaled 0 in counties without reentry locations, overall gonorrhea rates would decline by more than 63 cases (down from 99.0 to 35.8). It also shows that rates in county with the highest percentages of Black residents would decrease by more than 98 (down from 171.0 to 72.2). The gonorrhea rates in

county with the lowest percentages of Black residents would increase by 9.8 (up from 14.6 to 24.4). However, the gonorrhea rates in county with intermediate percentages of Black residents would fall by more than 24 cases (down from 48.4 to 23.9). If black isolation could be eliminated and reentry locations moved out of disproportionately black counties, it is estimated that the gap in gonorrhea rates between white counties and disproportionately black counties could be reduced from 84 cases to 48 cases per 100,000 residents. The next section will discuss limitations of the research.

### **Limitations**

This research does have some limitations. First, it cannot offer a pure sexual network *analysis* because the NSFG does not examine the size nor composition of the respondents' sexual networks. Second, because the individual-level data are cross-sectional, it is not possible to assess the temporal ordering of those sexual network-related factors and how they are related to STDs. This does not allow for a causal explanation. Moreover, the data do not provide a complete picture of factors related to STDs.

The individual data only include non-institutionalized individuals and may have missed those (e.g., the incarcerated) needed for accurate representation. There are concerns over reporting errors with respect to self-reported data of health status. Using data from the combined 1986-1994 cohorts of the National Health Interview Survey, McGee et al. (1999) found that self-reporting data on health status was reliable and related to mortality. Reporting whether one has been told they have an STD is a sensitive decision that can increase response error, and “[t]he results could be affected by under-reporting of sensitive behaviors; [however], using ACASI has been found to yield more complete reporting of these measures than other types of

questionnaires” (Chandra et al., 2012: 6). According to Ghanem et al. (2005), the use of Audio Computer Assisted Self Interview (ACASI) reduces errors in the reporting of sensitive information such as sexual behaviors and undesirable traits like having an STD. Because NSFG utilized the ACASI reporting system, self-reports should be more reliable (Harawa et al., 2003). Another limitation of the NSFG is that the age range of the NSFG is 15–44 years. Therefore, NSFG data cannot be used to estimate the HIV/ [STD] risk-related behavior of those under age 15 or over age 44 years, among whom these public health concerns are also relevant” (Chandra et al., 2012).

The county-level data also only allow for the examination of correlation rather than causation. It would be preferable to have data at a lower level of aggregation that still provides national coverage. Also, it is difficult to examine causal mechanisms using this dataset. Neither the individual-level or community-level dataset allow for the examination of homophobia, sexism, or other exogenous factors that may be related to racial disparities in STDs. It would be great to have characteristics of those who reentered a community after incarceration and to be able to examine these questions using multilevel analysis.

### **Discussion and Conclusions**

In the three substantive chapters of the dissertation, I have discussed racial disparities in STDs. I reviewed the sociological literature that attempts to explain these disparities. Each chapter presents the theoretical framework used to motivate the research questions and hypotheses. Each chapter also provides a justification for the use of the analytical framework, presents the models assessed, and presents the results and discussions. The dissertation utilizes both individual-level and community-level datasets to explore the relationship between racial



disparities in STDs and sexual network factors, residential segregation, and incarceration and ex-offender reentry locations.

Chapter 2 analyzed the relationship between the likelihood of reporting an STD for Black women compared with other race by gender groups with sexual network factors. The data from the NSFG is a national sample “of men and women 15-44 years of age living in households in the United States.” These data were used to test several hypotheses derived from the sexual networks literature. It used a series of logistic regression models to test these hypotheses. The findings suggests that when taking into consideration the baseline and sexual network factors, the STD gap between Black women and all other social locations except other race women becomes somewhat larger when baseline and sexual network factors are taken into consideration. The findings also suggest that, generally sexual network factors are not related to reporting STDs in ways that differ systematically by social location. Overall, sexual networks alone do not explain racial and gender disparities in STDs, and other factors need to be examined.

Chapter 3 utilized a dataset compiled from multiple sources that include county-level indicators that research has suggested is important to county rates of STDs. It examined the relationship between county rates of STDs and black isolation and white isolation. It asked whether racial residential segregation is related to community rates of chlamydia and gonorrhea. It also sought to determine how black isolation and white isolation are related to rates of chlamydia and gonorrhea and whether these relationships vary by racial composition of county. It tested hypotheses concerning black isolation and white isolation operate in different fashions in different community types with respect to county rates of STDs. The analysis is based on a series of Ordinary Least Squares (OLS) regression models in which community rates of

chlamydia and gonorrhea are the dependent variables. The analysis also examines how these factors are related to chlamydia rates and gonorrhea rates when stratified by racial composition of county. The stratified analyses provide some assessment of how great the disparities in STDs are between communities with the lowest percentages of Black residents and communities with the highest percentages of Black residents, net of other community health-related factors. The results showed that racial residential segregation is related to county rates of chlamydia and gonorrhea. Generally, black isolation is associated with increasing rates of chlamydia and gonorrhea and white isolation is related to decreasing rates of chlamydia and gonorrhea. However, in communities with the lowest percentages of Black residents, both black isolation and white isolation are associated with decreases in chlamydia, net of other factors. The patterns for gonorrhea are a bit more ambiguous, but clearly in such counties, black isolation does not have the same relationship to gonorrhea rates as it does in counties with higher percentages of Black residents. In counties with intermediary and the highest percentages of Black residents, black isolation is associated with increases in chlamydia rates and gonorrhea rates. The black county-white county chlamydia and gonorrhea rates disparities would be greatly reduced if black isolation were eliminated.

Chapter 4 drew on the same dataset used in Chapter 3; however, the central factors of concerns were county rates of incarceration and whether the county has a reentry location. The literature suggests that these factors may be related to county rates of STDs. The chapter sought to answer several questions: (1) Is incarceration related to community rates of chlamydia and gonorrhea? (2) How are lockup rates and reentry locations related to rates of chlamydia and gonorrhea? (3) Do these relationships vary by racial composition of county? The analysis tested

hypotheses derived from the literature. The analysis was based on a series of Ordinary Least Squares (OLS) regression models in which community rates of chlamydia and gonorrhea were the dependent variables. The central independent variables were lockup rate and reentry location. The stratified analyses provide some assessment of how great the disparities in STDs are between communities with the lowest percentages of black residents and communities with the highest percentages of black residents, net of other community health-related factors. Generally, there is little support that higher lockup rates are associated with higher STD rates. Lockup rates are not systematically related to chlamydia rates; moreover, they are negatively related to gonorrhea rates.

There is substantially more support that counties with reentry locations will have higher rates of chlamydia and gonorrhea. Counties with reentry locations do have significantly higher rates of chlamydia and gonorrhea. The stratified analysis showed that counties with reentry locations have higher chlamydia and gonorrhea rates except in counties with the lowest percentages of Black residents.

The results also suggest that if black isolation could be eliminated and reentry locations moved out of disproportionately black counties, it is estimated that the racial gaps in chlamydia and gonorrhea rates between white counties and disproportionately black counties could be reduced.

### **Implications and Future Directions**

The United States is facing an often invisible crisis of racial disparities in STDs (Eng and Butler, 1997). Scholars have put forth several compelling theories to help explain racial disparities in STDs. Sociologists, in particular, are concerned with those theories that move

beyond individual behavior and examine group-level and structural mechanisms that create and maintain these disparities. For example, sexual network theories examine how group level processes inhibit or exacerbate infection between and within groups. Factors such as having high-risk partners are related to reporting STDs. But such factors do not do much to account for the gaps between Black women and other race by gender groups.

Socioenvironmental theories examine factors such as residential segregation and incarceration. These factors play key roles in creating and maintaining STD disparities. Although scholars tend to examine the independent effect of any specific factor on STD disparities, this dissertation demonstrates that these factors are, indeed, related.

This research has helped identify some new factors that apparently help explain disparities in STDs. A remaining challenge for the nation, however, is to address these issues with effective policies that eliminate them. According to the initiatives of Healthy People 2020, eliminating racial disparities in STDs is a primary goal. The initiative calls for policies that would increase awareness disparities of STD rates, partner characteristics, and demographics. Adimora and Schoenbach (2005) argue that public health policies need to move beyond a singular focus on individual behaviors. They claim that STD prevention policies should include community-level interventions that tackle unemployment, incarceration, and lack of access to quality health care. All of these structural factors are related to community rates of chlamydia and gonorrhea.

Bratter and Gorman (2011) suggest that African Americans face disparities in health because of such structural factors. They assert that low SES factors affect African Americans as well as whites. However, African Americans face an increased burden of structural violence

beyond the effects of low SES on health outcomes. Addressing racial disparities in STDs requires a commitment to eliminating the structural level factors that maintain these disparities.

LaVeist et al. (2011) suggest that policies such as the Racial and Ethnic Approaches to Community Health (REACH) program would help to address lack of health care, food deserts, and improved educational opportunities. This program was instituted by CDC and operates in 40 communities within the US. It addresses a host of health problems plaguing African American communities such as diabetes, asthma, heart disease and cervical cancer. By partnering with businesses, REACH promotes healthy lifestyles and provides needed resources to those in these communities. Such policies can promote healthy behaviors, provide access to testing and treatment, and provide needed education that may reduce incarceration and recidivism rates, all of which would help reduce the disparity in STD rates (LaVeist et al., 2011).

Scholars like Farley (2006) assert that structural factors are intertwined and complex, making it difficult to provide any one policy that would eliminate the racial disparities in STDs. This does not mean that the cause is hopeless. For example, the U.S. government established programs such as the Syphilis Eradication Effort (CDC, 2006), which sought to completely eliminate syphilis. As a result, rates of syphilis dropped dramatically. This kind of effort should be extended to other STD reduction programs. However, “until these factors can be changed, STD control efforts will still have to rely on traditional methods of control, specifically clinical services, screening, partner notification, condom distribution, and promotion of safer behaviors by individuals” (Farley, 2006: S64).

Overall, this dissertation illustrates how powerfully group-level and structural factors are related to racial disparities in STDs rates. With respect to Black women, it demonstrates that

even when they have similar sexual network patterns and sexual behaviors as whites, they remain at greater risk. An intersectional approach demands an examination of the contextual factors that may lead Black men and women to remain at greater risk despite their individual efforts. Using structural explanations such as residential segregation and mass incarceration this research points to potential factors that may help explain the STD disparities between Black women and other social locations. This research extends the scholarly discussion of racial disparities in STDs. In doing so, it remains faithful to the tenets of the intersectionality framework by empirically testing the simultaneity of social location variables that may be related to disparities in STDs. The research also highlights the importance of moving beyond behavioral and sexual network factors to explain disparities in STDs between social location groups, and it demonstrates that sexual network factors overall do not operate qualitatively differently for Black women compared with other race by gender groups. It also calls for further examination of the role of contextual factors that may help explain race by gender disparities. In future research, it may be possible to embed factors from the individual-level analysis within a county-level framework using multi-level analysis.

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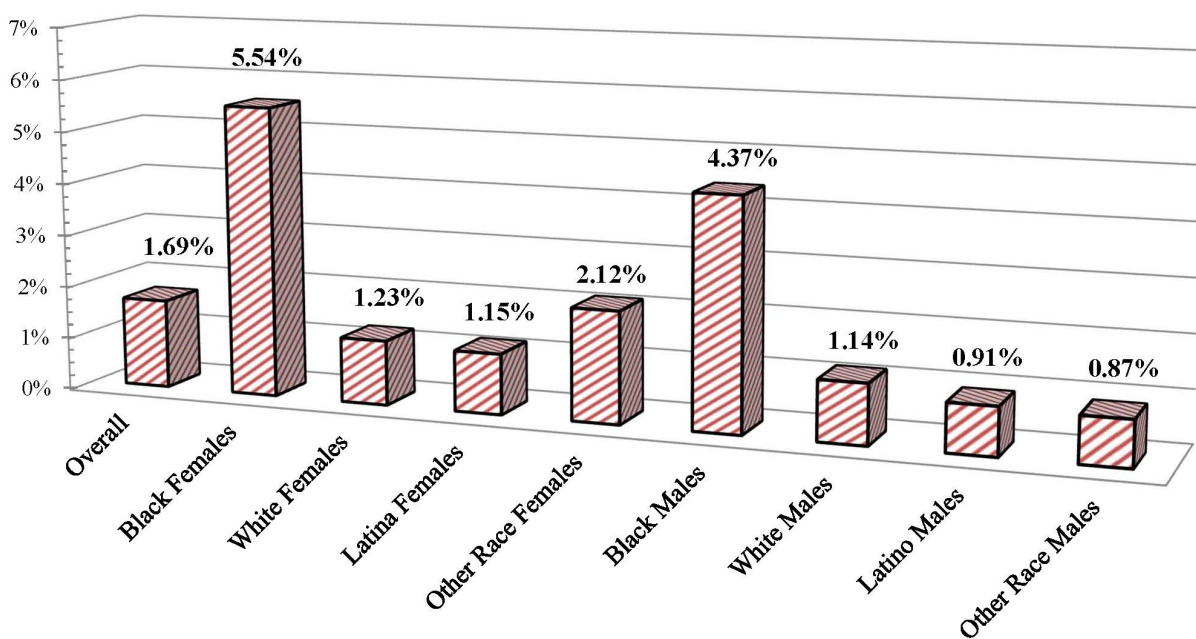
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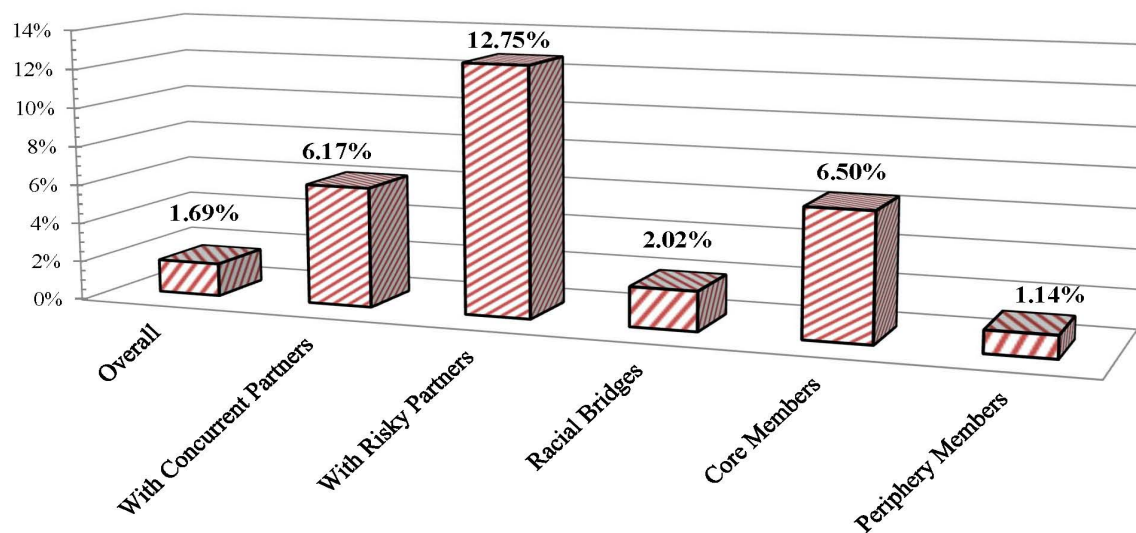
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## FIGURES AND CHARTS

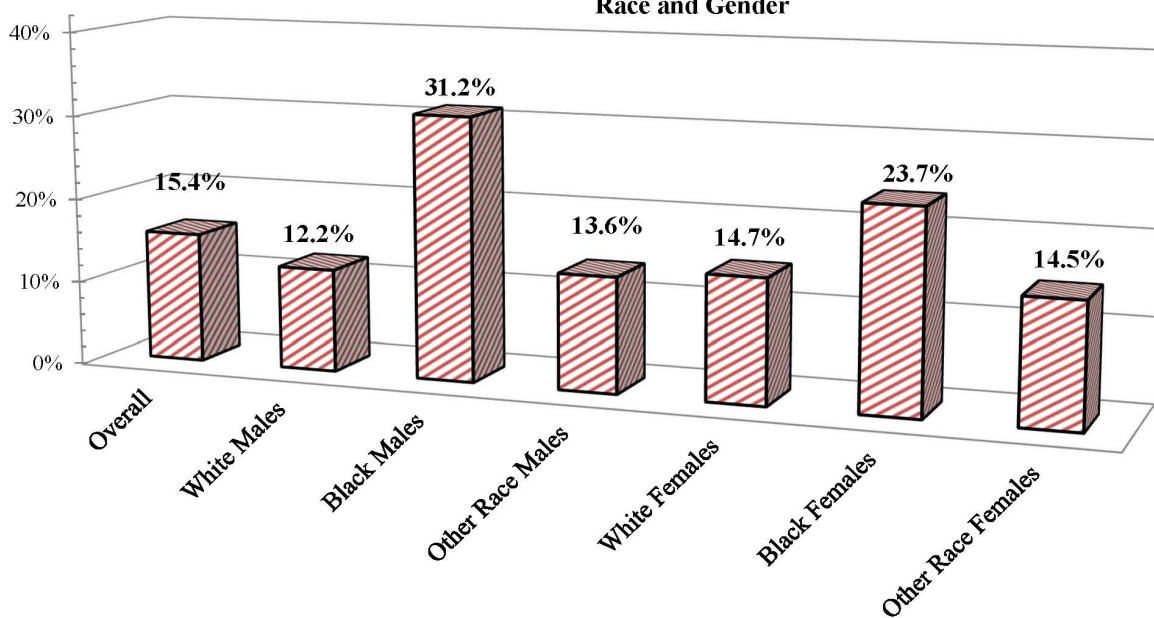
**Figure 2.1:**  
**Percentage Reporting an STD by Social Location**



Pearson Chi2 (7) = 244.3,  $p < .001$

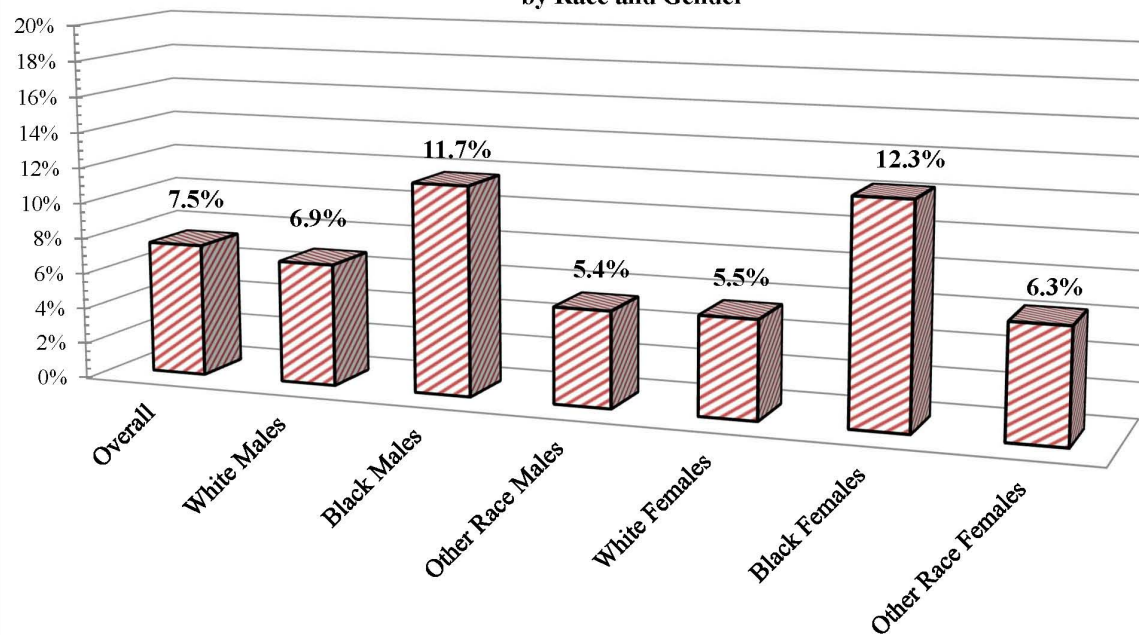
**Figure 2.2 Percentage of Respondents with STDs by Sexual Network Factors**

**Figure 2.3: Percentage of Respondents Tested for STDs within the Past Year by Race and Gender**



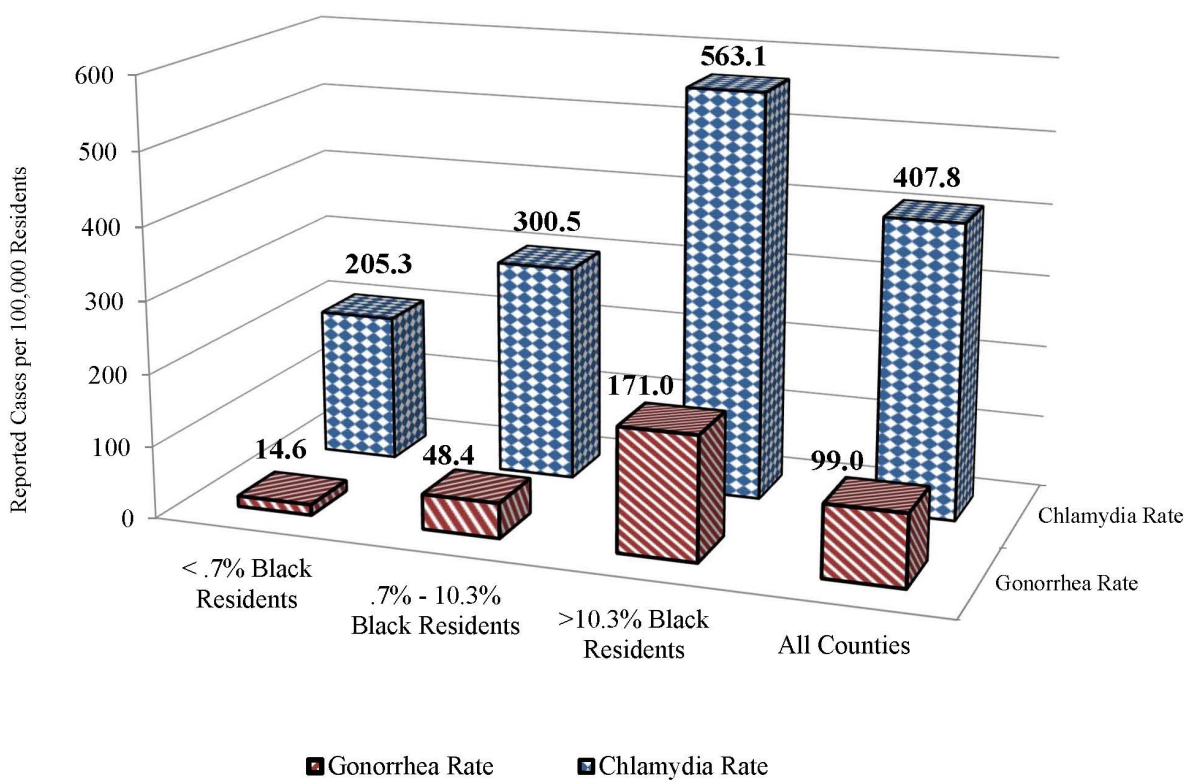
Pearson Chi2 (5) = 423.2, Pr = 0.000

**Figure 2.4: Percentage of Respondents Tested within the Past Year Reporting an STD by Race and Gender**



Pearson Chi2 (5) = 39.9, Pr = 0.006

**Figure 3.1:**  
**Chlamydia Rates and Gonorrhea Rates by Race of County**



**Figure 3.2:**  
**Predicted Chlamydia Rates and Gonorrhea Rates by Race of County**  
**if Black Isolation Equaled 0**

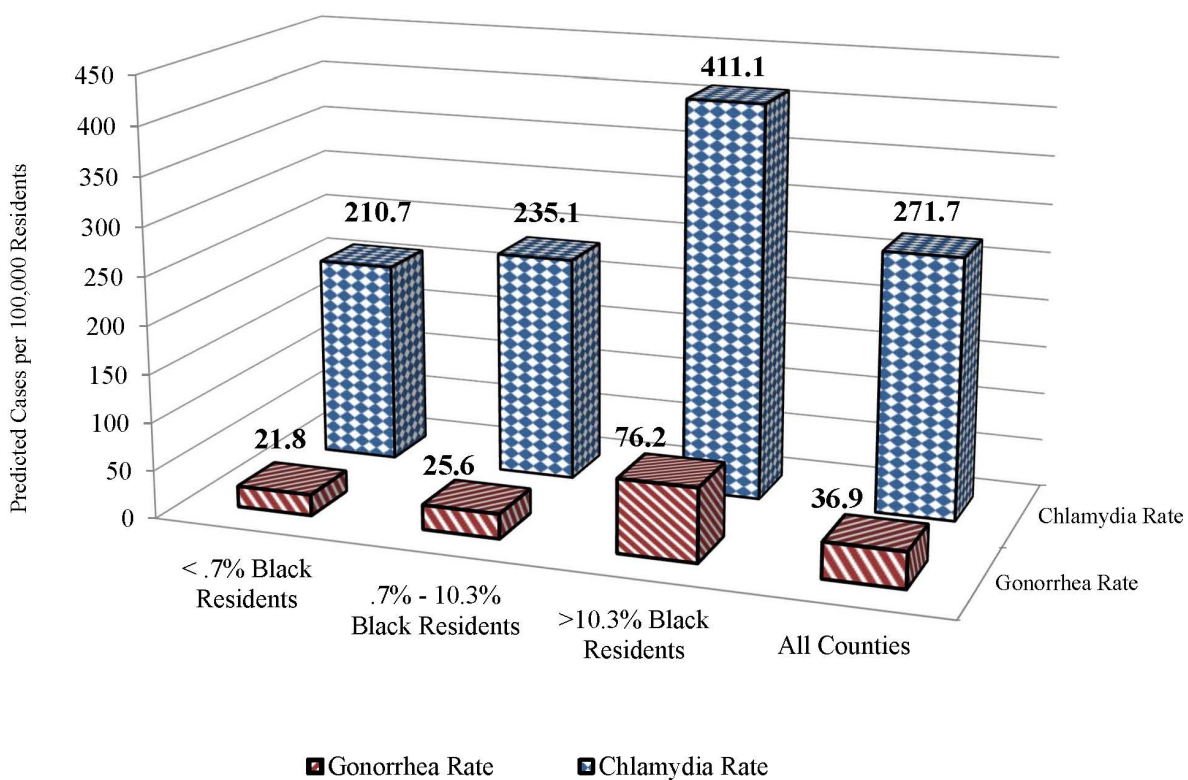


Figure 4.1

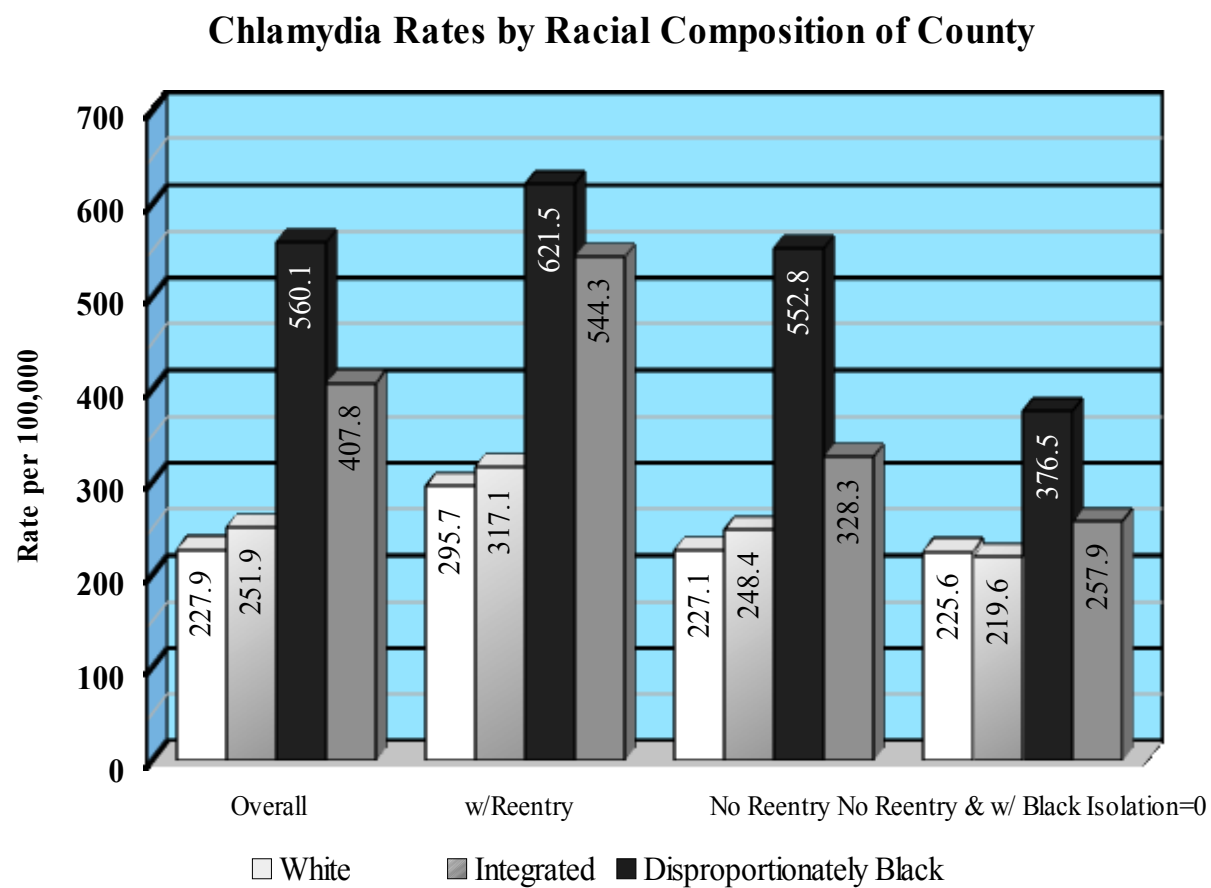
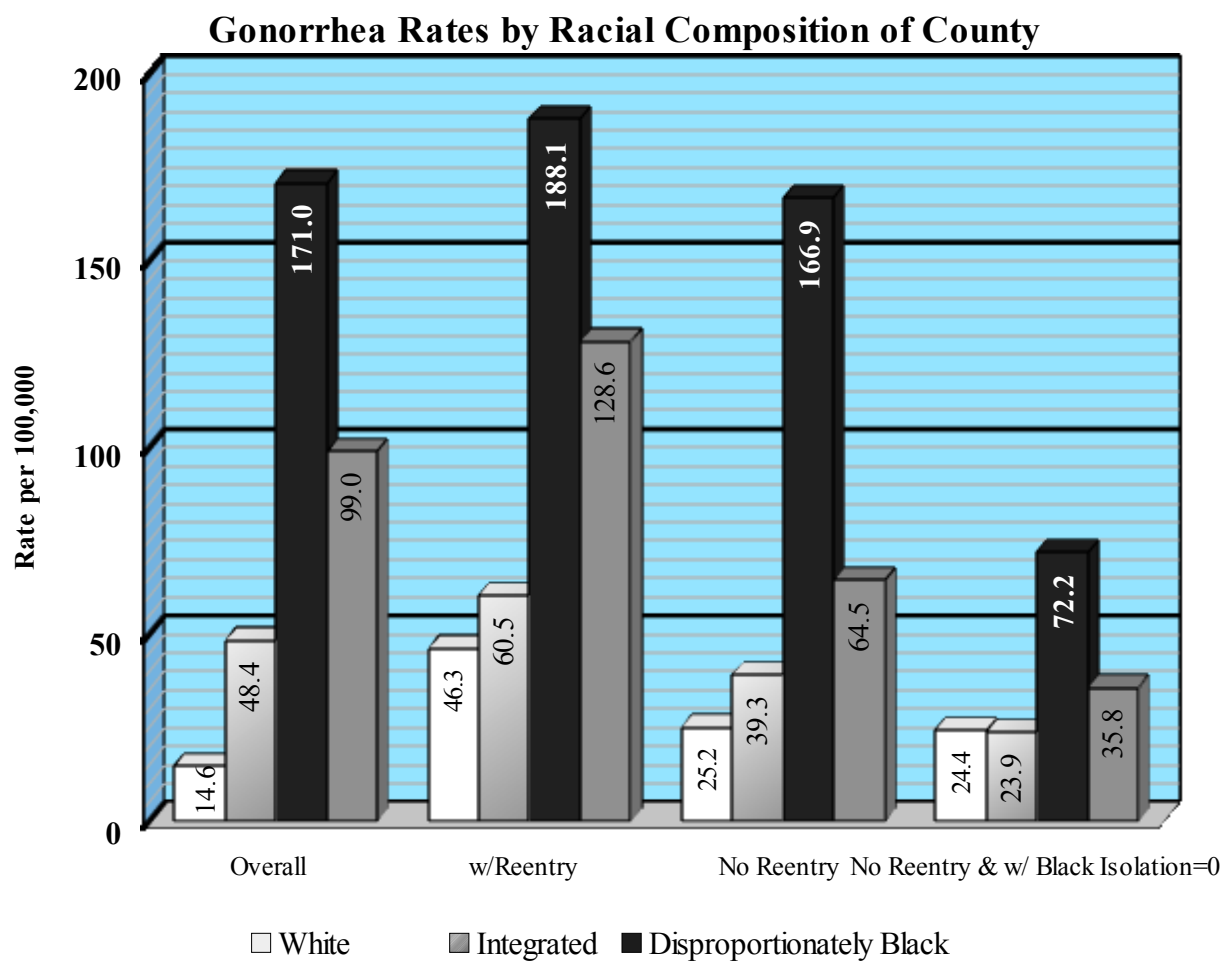




Figure 4.2



## TABLES

<b>Table 2.1:</b>			
<b>A Comparison of Respondents with STDs to Those Without STDs on Selected Characteristics</b>			
	<b>Without STDs</b>	<b>With an STD</b>	<b>Overall</b>
<b>Race by Gender</b>			
Black Female (n=2,535)	6.9%	23.6%	7.2%
White Female (n=6,301)	30.9%	22.4%	30.8%
Latina Female (n=2,723)	8.5%	5.8%	8.5%
Other Race Female (n=720)	3.4%	4.3%	3.4%
Black Male (n=1,854)	6.1%	16.2%	6.3%
White Male (n=5,448)	31.2%	20.9%	31.1%
Latino Male (n=2,409)	9.6%	5.2%	9.6%
Other Race Male (n=692)	3.3%	1.7%	3.2%
Total (n=22,682)	100.0%	100.0%	100.0%
Pearson: Uncorrected chi2(7) = 244.28, Design-based F(5.7, 551.1) = 18.07 Pr = 0.00			
<b>Sexual Network Variables</b>			
Concurrent Partner			
Not Concurrent (n=20,479)	90.9%	65.2%	90.5%
Concurrent (n=2,203)	9.1%	34.8%	9.5%
Total (n=22,682)	100.0%	100.0%	100.0%
Pearson Chi2(1) = 288.45 Design-based F(1.00 96.00) = 150.882 Pr = 0.000			
Riskiness of Partners			
Not Risky (n=19,865)	98.2%	86.4%	98.0%
Risky Partner(s) (n=2,817)	1.8%	13.6%	2.0%
Total (n=22,682)	100.0%	100.0%	100.0%
Pearson Chi2(1) = 249.8256 Design-based F(1.00 96.00) = 104.85 Pr = 0.000			
Racial Bridge Status			
Not a Bridge (n=17,674)	81.0%	77.2%	80.9%
Racial Bridge (n=4,903)	19.0%	22.8%	19.1%
Total (n=22,577)	100.0%	100.0%	100.0%
Pearson Chi2(1) = 3.3830 Design-based F(1.00 96.00) = 1.5605 Pr = 0.215			

<b>Table 2.1 (Cont.)</b>			
Core Status			
Not Core (n=21,474)	95.7%	82.5%	95.4%
Core (n=1,151)	4.3%	17.5%	4.6%
Total (n=22,625)	100.0%	100.0%	100.0%
Pearson Chi2(1) = 150.184 Design-based F(1.00 96.00) = 65.5050 Pr = 0.000			
Periphery Status			
Not Periphery (n=4,061)	14.6%	42.6%	15.1%
Periphery (n=18,564)	85.4%	57.4%	84.9%
Total (n=22,625)	100.0%	100.0%	100.0%
Pearson Chi2(1) = 229.165 Design-based F(1.00 96.00) = 92.2663 Pr = 0.000			
Age			
< 20 (n=4,662)	17.2%	16.4%	17.2%
20-34 (n=11,606)	48.7%	55.7%	48.8%
35+ (n=6,414)	34.1%	27.9%	34.0%
Total (n=22,682)	100.0%	100.0%	100.0%
Pearson Chi2(2) = 8.287 Design-based F(1.91 183.63) = 2.4404 Pr = 0.092			
Education			
< HS (n=6,924)	26.9%	36.7%	27.1%
HS (n=5,475)	23.4%	28.3%	23.5%
> HS (n=10,283)	49.7%	34.9%	49.5%
Total (n=22,682)	100.0%	100.0%	100.0%
Pearson Chi2(2) = 34.042 Design-based F(1.97 189.31) = 9.1623 Pr = 0.000			
Income			
<\$20K (n=6,246)	21.5%	42.4%	21.8%
\$20-40K (n=6,288)	26.1%	21.3%	26.1%
\$40-60K (n=4,049)	18.3%	18.5%	18.3%
\$60K (n=6,099)	34.1%	17.9%	33.8%
Total (n=22,682)	100.0%	100.0%	100.0%
Pearson Chi2(3) = 107.93 Design-based F(2.81 269.67) = 14.9002 Pr = 0.000			
Marital Status			
Married (n=6,795)	39.8%	23.5%	39.5%
Cohabiting (n=2,535)	11.6%	16.3%	11.7%
Never Married (n=11,368)	41.4%	51.4%	41.6%

<b>Table 2.1 (Cont.)</b>			
Pearson Chi2(1) = 9.4954 Design-based F(1.00 96.00) = 3.9896 Pr = 0.049			
Age at First Sex			
Never had Sex (n=3,553)	14.7%	4.5%	14.5%
Before Age 16 (n=6,225)	25.4%	47.5%	25.7%
16-18 (n=8,393)	38.5%	38.1%	38.5%
After 18 (n=4,376)	21.5%	9.8%	21.3%
Total (n=22,547)	100.0%	100.0%	100.0%
Pearson Chi2(3) = 121.54 Design-based F(2.76 265.20) = 22.6470 Pr = 0.000			
Same Sex Intercourse			
No Same Sex Intercourse (n=20,037)	90.9%	72.2%	90.6%
Same Sex Intercourse (n=2,453)	9.1%	27.8%	9.4%
Total (n=22,490)	100.0%	100.0%	100.0%
Pearson Chi2(1) = 154.64 Design-based F(1.00 96.00) = 77.6441 Pr = 0.000			
Used Condom?			
Did Not Use Condom (n=12,340)	59.0%	65.4%	59.1%
Used Condom (n=6,773)	26.7%	31.3%	26.8%
Never had Sex (n=3,448)	14.3%	3.3%	14.1%
Total (n=22,561)	100.0%	100.0%	100.0%
Pearson Chi2(2) = 38.064 Design-based F(1.94 186.47) = 13.3182 Pr = 0.000			
Used Drugs			
Did Not Use Drugs (n=17,337)	78.8%	60.8%	78.5%
Used Drugs (n=5,345)	21.2%	39.2%	21.5%
Total (n=22,682)	100.0%	100.0%	100.0%
Pearson Chi2(1) = 71.8709 Design-based F(1.00 96.00) = 45.9095 Pr = 0.000			
Anal Sex			
No Anal Sex (n=15,106)	65.5%	47.1%	65.2%
Has Anal Sex (n=7,576)	34.5%	52.9%	34.8%
Total (n=22,682)	100.0%	100.0%	100.0%
Pearson Chi2(1) = 56.5923 Design-based F(1.00 96.00) = 27.1683 Pr = 0.000			

**Table 2.2**

**Predicting the Likelihood of Reporting an STD with Social Location,  
Net of Baseline Factors and Sexual Network Factors**

Independent Variables	Model 1		Model 2		Model 3	
	Coefficient	Odds Ratios	Coefficient	Odds Ratios	Coefficient	Odds Ratios
White Females	-1.551***	.212***	-1.383***	.251***	-1.399***	.247***
(S.E.)	(.184)	(.039)	(.197)	(.049)	(.198)	(.049)
Latina Females	-1.617***	.199***	-1.399***	.247***	-1.524***	.218***
(S.E.)	(.234)	(.046)	(.315)	(.078)	(.289)	(.063)
Other Race Females	-.994*	.370*	-.447	.64	-.436	.647
(S.E.)	(.444)	(.164)	(.476)	(.305)	(.456)	(.295)
Black Males	-.249	.78	-.122	.885	-.434	.648
(S.E.)	(.198)	(.154)	(.207)	(.183)	(.243)	(.157)
White Males	-1.630***	.196***	-1.257***	.284***	-1.311***	.270***
(S.E.)	(.232)	(.045)	(.252)	(.072)	(.250)	(.067)
Latino Males	-1.848***	.157***	-1.753***	.173***	-1.988***	.137***
(S.E.)	(.278)	(.044)	(.362)	(.063)	(.356)	(.049)
Other Race Males	-1.894**	.151**	-1.217*	.296*	-1.565**	.209**
(S.E.)	(.627)	(.094)	(.590)	(.174)	(.555)	(.116)
Education			-.108**	.897**	-.104**	.901**
(S.E.)			(.036)	(.032)	(.037)	(.033)
Income			-.106**	.900**	-.100**	.905**
(S.E.)			(.039)	(.035)	(.038)	(.034)
Age			-.013	.987	-.009	.991
(S.E.)			(.009)	(.009)	(.010)	(.010)
Immigrant			-.127	.88	-.166	.847
(S.E.)			(.418)	(.368)	(.400)	(.339)
Married			-.285	.752	.053	1.054
(S.E.)			(.241)	(.181)	(.259)	(.273)
Cohabiting			-.077	.926	.203	1.225
(S.E.)			(.205)	(.189)	(.235)	(.288)
Age at First Sex			-.230*	.795*	-.162	.85
(S.E.)			(.103)	(.082)	(.106)	(.090)
				2.456**		2.187**
Same Sex			.898***	*	.783***	*
(S.E.)			(.203)	(.497)	(.212)	(.464)
Condom			-.206	.814	-.21	.811
(S.E.)			(.165)	(.134)	(.167)	(.135)

**Table 2.2 (Cont.)**

Drug Use			.271	1.311	.013	1.013
(S.E.)			(.175)	(.229)	(.177)	(.179)
Anal Sex			.467**	1.594**	.271	1.311
(S.E.)			(.155)	(.247)	(.163)	(.214)
Concurrent Partner					.625**	1.868**
(S.E.)					(.229)	(.428)
Risky Partner					.981**	2.666**
(S.E.)					(.316)	(.843)
Core Member					.317	1.373
(S.E.)					(.252)	(.345)
Periphery Member					-.497*	.608*
(S.E.)					(.243)	(.148)
Bridge					.205	1.228
(S.E.)					(.206)	(.253)
Constant	-2.837***		-.576		-.694	
(S.E.)	(.112)		(.516)		(.565)	
N	18,837	18,837	18,837	18,837	18,837	18,837

\*  $p < .05$     \*\*  $p < .01$     \*\*\*  $p < .001$

**Table 2.3**  
**Predicting the Likelihood of Reporting an STD with Social Location, Net of Baseline Factors, Sexual Network Factors, and Social Location by Sexual Network Interactions**

Independent Variables	Model 1			Model 2			Model 3			Model 4			Model 5		
	Concurrent Partners			Risky Partners			Core Member			Periphery Member			Racial Bridge		
	Coefficient	Ratios	Odds	Coefficient	Ratios	Odds	Coefficient	Ratios	Odds	Coefficient	Ratios	Odds	Coefficient	Ratios	Odds
White Females	-1.408***	.245***		-1.451***	.234***		-1.421***	.241***		-1.568***	.208***		-1.469***	.230***	
(S.E.)	(.236)	(.058)		(.211)	(.049)		(.217)	(.052)		(.269)	(.056)		(.229)	(.053)	
Latina Females	-1.826***	.161***		-1.819***	.162***		-1.778***	.169***		-1.111*	.329*		-1.394***	.248***	
(S.E.)	(.312)	(.050)		(.267)	(.043)		(.251)	(.043)		(.425)	(.140)		(.366)	(.091)	
Other Race Females	-.753	.471		-.48	.619		-.451	.637		-2.063	.127		-1.329*	.265*	
(S.E.)	(.629)	(.296)		(.476)	(.294)		(.484)	(.309)		(1.069)	(.136)		(.522)	(.138)	
Black Males	-.529	.589		-.506	.603		-.403	.669		-.674	.51		-.265	.767	
(S.E.)	(.309)	(.182)		(.276)	(.166)		(.271)	(.181)		(.341)	(.174)		(.266)	(.204)	
White Males	-1.191***	.304***		-1.285***	.277***		-1.373***	.253***		-1.296***	.274***		-1.374***	.253***	
(S.E.)	(.292)	(.089)		(.252)	(.070)		(.266)	(.067)		(.330)	(.090)		(.247)	(.062)	
Latino Males	-2.065***	.127***		-1.946***	.143***		-1.867***	.155***		-2.274***	.103***		-2.011***	.134***	
(S.E.)	(.486)	(.062)		(.400)	(.057)		(.381)	(.059)		(.502)	(.052)		(.515)	(.069)	
Other Race Males	-1.304	.271		-1.667**	.189**		-1.467*	.231*		-3.201**	.041**		-2.612***	.073***	
(S.E.)	(.686)	(.186)		(.598)	(.113)		(.677)	(.156)		(1.104)	(.045)		(.671)	(.049)	
Education	-.105**	.900**		-.107**	.898**		-.104**	.901**		-.107**	.899**		-.107**	.898**	
(S.E.)	(.037)	(.033)		(.037)	(.033)		(.038)	(.034)		(.037)	(.034)		(.037)	(.033)	
Income	-.102**	.903**		-.099*	.906*		-.102**	.903**		-.101**	.904**		-.098*	.907*	
(S.E.)	(.038)	(.034)		(.039)	(.035)		(.038)	(.034)		(.037)	(.034)		(.038)	(.035)	
Age	-.008	.992		-.008	.992		-.009	.991		-.009	.991		-.009	.991	
(S.E.)	(.010)	(.010)		(.010)	(.010)		(.010)	(.010)		(.010)	(.010)		(.010)	(.010)	
Immigrant	-.101	.904		-.162	.85		-.151	.86		-.147	.864		-.145	.865	

Table 2.3 (Cont.)

(S.E.)	(.411)	(.371)	(.404)	(.344)	(.379)	(.326)	(.391)	(.338)	(.408)	(.353)
Married	.045	1.046	.041	1.041	.07	1.072	.057	1.059	.072	1.074
(S.E.)	(.258)	(.270)	(.261)	(.272)	(.266)	(.285)	(.264)	(.279)	(.261)	(.280)
Cohabiting	.2	1.221	.203	1.226	.215	1.24	.208	1.231	.209	1.232
(S.E.)	(.236)	(.288)	(.235)	(.288)	(.235)	(.291)	(.236)	(.291)	(.232)	(.286)
Age at First Sex	-.164	.849	-.157	.855	-.155	.856	-.155	.856	-.148	.862
(S.E.)	(.109)	(.092)	(.111)	(.095)	(.106)	(.091)	(.108)	(.092)	(.103)	(.089)
Same Sex	.764***	2.146***	.764***	2.148***	.785***	2.192***	.784***	2.190***	.762***	2.142***
(S.E.)	(.214)	(.459)	(.211)	(.454)	(.215)	(.471)	(.217)	(.476)	(.214)	(.458)
Condom	-.226	.797	-.220	.802	-.228	.796	-.217	.805	-.206	.814
(S.E.)	(.165)	(.131)	(.168)	(.135)	(.170)	(.135)	(.167)	(.135)	(.166)	(.135)
Drug Use	.029	1.03	.003	1.003	.009	1.009	.014	1.015	.030	1.030
(S.E.)	(.176)	(.181)	(.179)	(.179)	(.179)	(.181)	(.179)	(.181)	(.175)	(.181)
Anal Sex	.262	1.299	.274	1.315	.275	1.317	.277	1.319	.273	1.314
(S.E.)	(.164)	(.213)	(.163)	(.215)	(.163)	(.215)	(.163)	(.214)	(.164)	(.216)
Concurrent Partner	.563*	1.757*	.625**	1.867**	.619**	1.857**	.624**	1.867**	.625**	1.868**
(S.E.)	(.283)	(.498)	(.231)	(.431)	(.232)	(.431)	(.232)	(.433)	(.226)	(.423)
Risky Partner	.979**	2.662**	.436	1.547	.993**	2.700**	.972**	2.642**	.972**	2.644**
(S.E.)	(.319)	(.851)	(.476)	(.737)	(.303)	(.818)	(.320)	(.846)	(.317)	(.838)
Core Member	.339	1.404	.307	1.359	.039	1.039	.344	1.41	.356	1.428
(S.E.)	(.255)	(.358)	(.250)	(.340)	(.554)	(.576)	(.266)	(.375)	(.247)	(.353)
Periphery Member	-.515*	.598*	-.514*	.598*	-.495*	.610*	-.678*	.508*	-.491*	.612*
(S.E.)	(.239)	(.143)	(.245)	(.146)	(.242)	(.148)	(.284)	(.144)	(.243)	(.149)
Bridge	.198	1.219	.199	1.22	.204	1.227	.211	1.235	-.07	.932
(S.E.)	(.207)	(.253)	(.208)	(.253)	(.202)	(.248)	(.204)	(.253)	(.621)	(.579)
White Female X Sex. Network	.042	1.043	.722	2.06	.267	1.306	.263	1.301	.556	1.743
(S.E.)	(.435)	(.454)	(.658)	(1.354)	(.716)	(.935)	(.361)	(.470)	(.799)	(1.394)





Table 2.4:  
Predicted Probabilities of Reporting an STD for Social Location by Sexual Network Interactions

Social Location	Model 1		Model 2		Model 3		Model 4		Model 5	
	Concurrent Partner	Not Concurrent	Risky Partner	No Risky Partner	Core Member	Not Core Member	Peripheral Member	Not Peripheral Member	Racial Bridge	Not Racial Bridge
Black Women	0.0642	0.0379	0.0607	0.0408	0.0413	0.0406	0.0367	0.0697	0.0442	0.0395
White Women	0.0078	0.0045	0.0071	0.0046	0.0049	0.0047	0.0037	0.0072	0.0055	0.0045
Latina Women	<b>0.0043*</b>	0.0025	<b>0.0041*</b>	0.0026	<b>0.0029*</b>	0.0028	0.0043	0.0084	0.0032	0.0026
Other Race Women	0.0103	0.0059	0.0125	0.0081	0.0087	0.0084	0.0015	0.003	0.0098	0.008
Black Men	0.0131	0.0075	0.0125	0.0081	0.0093	0.009	0.0061	0.0119	0.0105	0.0086
White Men	0.0086	0.0049	0.0074	0.0048	0.0047	0.0046	0.0042	0.0082	0.0053	0.0043
Latino Men	0.0034	0.0019	0.0035	0.0023	0.0026	0.0025	0.0015	0.0029	0.0029	0.0023
Other Race Men	0.006	0.0034	0.0039	0.0026	0.0033	0.0031	0.0005	0.001	<b>0.0037*</b>	0.003

Table 2.5

Predicting the Likelihood of Reporting an STD Among Those Tested Within the Past Year with Social Location,  
Net of Baseline Factors, Sexual Network Factors, and Social Location by Sexual Network Interactions

Independent Variables	Model 1		Model 2		Model 3		Model 4		Model 5	
	Concurrent Partners		Risky Partners		Core Member		Periphery Member		Racial Bridge	
	Coefficient	Odds Ratios	Coefficient	Odds Ratios	Coefficient	Odds Ratios	Coefficient	Odds Ratios	Coefficient	Odds Ratios
White Females	-1.081***	.339***	-1.102***	.332***	-1.029***	.357***	-1.041**	.353**	-1.095***	.335***
(S.E.)	(.317)	(.108)	(.261)	(.087)	(.267)	(.095)	(.322)	(.114)	(.268)	(.090)
Latina Females	-1.590***	.204***	-1.976***	.139***	-1.732***	.177***	-.587	.556	-1.813**	.163**
(S.E.)	(.411)	(.084)	(.365)	(.051)	(.335)	(.059)	(.452)	(.251)	(.541)	(.088)
Other Race Females	.117	1.125	.157	1.170	.317	1.373	-1.795	.166	-1.823***	.161***
(S.E.)	(.948)	(1.066)	(.690)	(.807)	(.669)	(.919)	(1.141)	(.190)	(.474)	(.077)
Black Males	-.342	.710	-.336	.714	-.257	.774	-.563	.569	-.246	.782
(S.E.)	(.426)	(.302)	(.326)	(.233)	(.339)	(.263)	(.368)	(.210)	(.339)	(.265)
White Males	-.385	.681	-.497	.609	-.626	.535	-.506	.603	-.685*	.504*
(S.E.)	(.352)	(.240)	(.283)	(.172)	(.317)	(.170)	(.404)	(.244)	(.288)	(.145)
Latino Males	-1.032	.356	-1.145*	.318*	-.912*	.402*	-1.683**	.186**	-.995	.370
(S.E.)	(.590)	(.210)	(.522)	(.166)	(.457)	(.184)	(.537)	(.100)	(.555)	(.205)
Other Race Males	-.191	.826	-.543	.581	-.572	.564	-2.134	.118	-2.755***	.064***
(S.E.)	(.703)	(.581)	(.620)	(.360)	(.668)	(.377)	(1.124)	(.133)	(.629)	(.040)
Education	-.110*	.896*	-.116*	.891*	-.110*	.896*	-.117*	.889*	-.114*	.892*
(S.E.)	(.052)	(.047)	(.053)	(.047)	(.052)	(.046)	(.051)	(.045)	(.053)	(.047)
Income	-.100*	.905*	-.100*	.905*	-.103*	.902*	-.101*	.904*	-.097*	.907*
(S.E.)	(.043)	(.039)	(.043)	(.039)	(.043)	(.039)	(.043)	(.039)	(.043)	(.039)
Age	-.023	.978	-.021	.979	-.022	.978	-.021	.979	-.024	.977

Table 2.5 (Cont.)

(S.E.)	(.017)	(.017)	(.018)	(.017)	(.017)	(.017)	(.017)	(.017)	(.017)	(.017)	(.017)	(.017)
Immigrant	.073	1.075	.048	1.049	.073	1.076	.013	1.013	.078	1.082	(.017)	(.017)
(S.E.)	(.415)	(.446)	(.421)	(.442)	(.378)	(.406)	(.393)	(.398)	(.410)	(.444)	(.410)	(.444)
Married	.372	1.451	.367	1.443	.372	1.451	.360	1.434	.433	1.541	.433	1.541
(S.E.)	(.315)	(.457)	(.333)	(.481)	(.326)	(.474)	(.318)	(.457)	(.321)	(.495)	(.321)	(.495)
Cohabiting	.310	1.363	.267	1.306	.291	1.338	.301	1.351	.305	1.357	.305	1.357
(S.E.)	(.247)	(.337)	(.248)	(.324)	(.253)	(.338)	(.255)	(.344)	(.240)	(.325)	(.240)	(.325)
Age at First Sex	.012	1.012	.010	1.011	.026	1.027	.036	1.037	.045	1.046	.045	1.046
(S.E.)	(.146)	(.148)	(.150)	(.152)	(.140)	(.144)	(.139)	(.144)	(.137)	(.143)	(.137)	(.143)
Same Sex	.510*	1.665*	.522*	1.686*	.517*	1.677*	.495	1.641	.535*	1.707*	.535*	1.707*
(S.E.)	(.241)	(.401)	(.233)	(.392)	(.246)	(.412)	(.257)	(.422)	(.246)	(.421)	(.246)	(.421)
Condom	-.216	.806	-.245	.783	-.235	.790	-.221	.801	-.211	.810	-.211	.810
(S.E.)	(.189)	(.152)	(.202)	(.158)	(.206)	(.163)	(.196)	(.157)	(.190)	(.154)	(.190)	(.154)
Drug Use	.197	1.217	.201	1.223	.198	1.219	.184	1.202	.241	1.272	.241	1.272
(S.E.)	(.232)	(.283)	(.234)	(.285)	(.233)	(.284)	(.233)	(.280)	(.232)	(.295)	(.232)	(.295)
Anal Sex	-.167	.846	-.163	.850	-.151	.860	-.106	.900	-.142	.867	-.142	.867
(S.E.)	(.208)	(.176)	(.203)	(.172)	(.204)	(.176)	(.203)	(.183)	(.209)	(.182)	(.209)	(.182)
Concurrent Partner	.599	1.821	.568	1.764	.575	1.777	.592*	1.807*	.568	1.764	.568	1.764
(S.E.)	(.472)	(.859)	(.297)	(.524)	(.294)	(.522)	(.290)	(.524)	(.292)	(.516)	(.292)	(.516)
Risky Partner	.945*	2.572*	.091	1.095	.928**	2.529**	.927*	2.526*	.932*	2.539*	.932*	2.539*
(S.E.)	(.370)	(.951)	(.575)	(.630)	(.347)	(.877)	(.379)	(.959)	(.368)	(.936)	(.368)	(.936)
Core Member	.358	1.43	.324	1.383	.086	1.090	.343	1.409	.370	1.447	.370	1.447
(S.E.)	(.273)	(.390)	(.274)	(.379)	(.676)	(.737)	(.284)	(.400)	(.271)	(.391)	(.271)	(.391)
Periphery Member	-.326	.722	-.318	.727	-.290	.748	-.505	.603	-.311	.733	-.311	.733
(S.E.)	(.293)	(.212)	(.301)	(.219)	(.297)	(.222)	(.432)	(.261)	(.303)	(.222)	(.303)	(.222)
Bridge	.145	1.156	.148	1.159	.136	1.146	.151	1.163	-.640	.527	-.640	.527
(S.E.)	(.257)	(.297)	(.248)	(.287)	(.249)	(.285)	(.250)	(.291)	(.723)	(.381)	(.723)	(.381)



**Table 3.1:**  
**Selected Characteristics of Communities by Racial Composition of County**

Variables	< .7% Black Residents	.7% - 10.3% Black Residents	>10.3% Black Residents	All Counties
Chlamydia Rate (per 100,000)	205.3***	300.5***	563.1	407.8
Gonorrhea Rate (per 100,000)	14.6***	48.4***	171.0	99.0
Black Isolation Index Score	0.7***	12.4***	47.9	26.7
White Isolation Index Score	83.6***	80.3***	73.1	77.4
% Northeast	4.1***	13.5***	16.7	14.4
% Midwest	44.3***	27.1***	22.8	26.1
% South	22.8***	22.5***	55.7	36.7
% West	28.7***	36.9***	4.8	22.9
% College Graduates	18.0***	22.8***	21.1	21.8
% Latino	15.0	16.8***	14.3	15.7
Sex Ratio (Men per 100 Women)	99.2***	98.1***	94.7	96.7
% Unemployed	7.4***	7.5***	8.7	8.0
Median Income	42,105***	54,920***	50,216	52,313
% Immigrants	9.4	10.9***	16.5	13.2
% with Health Professional Shortage	16.4***	3.6	4.2	4.4
Population Density	76.5***	257.1***	197.6	223.3
Gini Index Score	42.7***	43.4***	45.8	44.4
N	808	1527	754	3089

\* p < .05, \*\* p < .01, \*\*\* p < .001

**Table 3.2:**  
**OLS Regression Models Predicting Chlamydia Rates with Black Isolation and White Isolation by Racial Composition of County**

Independent Variables	Chlamydia			
	Model 1	Model 2	Model 3	Model 4
	All Counties	< .7% Black	.7-10.3% Black	>10.3% Black
Black Isolation	6.207*** (0.492)	-1.108 (5.146)	3.036*** (0.582)	7.412*** (0.998)
White Isolation	-3.962*** (0.811)	-4.104*** (0.585)	-5.335*** (0.459)	-1.839 (2.535)
Constant	543.2*** (68.370)	549.0*** (56.290)	691.6*** (43.020)	332.9 (206.600)
N	3089	808	1527	754
R <sup>2</sup>	0.528***	0.333***	0.444***	0.311***
AIC	13.02	12.86	12.19	13.52
BIC	15433.34	5009.59	7437.02	5216.35

\* p<0.05, \*\* p<0.01, \*\*\* p<0.001

**Table 3.3:**

**OLS Regression Models Predicting Chlamydia Rates with Black Isolation and White Isolation by  
Racial Composition of County, Net of Other Community Factors**

Independent Variables	Chlamydia			
	Model 1 All Counties	Model 2 < .7% Black White Counties	Model 3 .7-10.3% Black Integrated Counties	Model 4 > 10.3% Black Black Counties
Black Isolation	4.315*** (0.488)	-18.24* (7.151)	3.119*** (0.450)	3.460*** (0.888)
White Isolation	-6.496*** (0.638)	-17.17*** (2.288)	-6.204*** (0.684)	-3.804** (1.243)
College Graduates	-0.67 (0.721)	0.112 (0.696)	0.0548 (0.577)	0.119 (1.258)
Percent Latino	-2.933** (0.988)	-12.69*** (2.099)	-0.00871 (0.686)	-3.804 (2.419)
Sex Ratio	-2.286 (1.462)	-0.0247 (1.674)	-0.398 (1.076)	-5.172 (2.864)
Percent Unemployed	10.94* (4.338)	1.497 (2.453)	-6.890* (2.971)	22.66** (7.145)
Median Income	-38.28*** (6.507)	14.52 (9.077)	-41.05*** (5.009)	-60.64*** (13.300)
Percent Immigrant	0.173 (0.120)	-0.271* (0.131)	0.282 (0.163)	-0.0515 (0.096)
Health Professional Shortage	-47.63*** (13.240)	(14.660) (12.060)	-54.34*** (11.940)	-63.31* (30.520)
Population Density	0.0559* (0.027)	0.275* (0.121)	0.0724** (0.025)	-0.0145 (0.055)
Gini Index	4.944* (2.370)	-2.073 (1.787)	-1.591 (1.577)	9.066** (3.359)
Constant	951.8*** (225.100)	1843.9*** (308.300)	1125.1*** (197.600)	906.0* (361.700)
N	3089	808	1527	754
R <sup>2</sup>	0.625***	0.649***	0.587***	0.538***
AIC	12.79	12.25	11.91	13.14
BIC	14757.45	4541.40	7059.78	4969.36

\* p< .05, \*\* p< .01, \*\*\* p< .001



**Table 3.4:**  
**OLS Regression Models Predicting Gonorrhea Rates with Black Isolation and White Isolation by**  
**Racial Composition of County**

Independent Variables	Gonorrhea			
	Model 1 All Counties	Model 2 < .7% Black White Counties	Model 3 .7-10.3% Black Integrated Counties	Model 4 > 10.3% Black Black Counties
Black Isolation	3.113*** (0.193)	-0.987 (1.244)	1.913*** (0.247)	3.310*** (0.369)
White Isolation	-0.289 (0.266)	-0.423** (0.158)	-0.706*** (0.211)	0.392 (0.721)
Constant	36.03 (22.870)	50.61*** (15.020)	81.50*** (18.310)	-19.55 (57.260)
N	3089	808	1527	754
R <sup>2</sup>	0.599***	0.060***	0.376***	0.348***
AIC	11.04	10.33	9.90	11.65
BIC	9296.77	2955.12	3937.12	3804.81

\* p< .05, \*\* p< .01, \*\*\* p< .001

**Table 3.5:**  
**OLS Regression Models Predicting Gonorrhea Rates with Black Isolation and White Isolation**  
**by Racial Composition of County, Net of Other Community Factors**

Independent Variables	Gonorrhea			
	Model 1 All Counties	Model 2 < .7% Black White Counties	Model 3 .7-10.3% Black Integrated Counties	Model 4 > 10.3% Black Black Counties
Black Isolation	2.448*** (0.210)	-4.511 (2.428)	1.969*** (0.212)	2.017*** (0.352)
White Isolation	-1.797*** (0.320)	-3.004* (1.371)	-1.369** (0.445)	-0.92 (0.498)
College Graduates	-0.500* (0.244)	-0.259 (0.157)	-0.249 (0.192)	-0.178 (0.434)
Percent Latino	-1.585*** (0.337)	-2.504* (1.201)	-0.287 (0.474)	-2.180*** (0.471)
Sex Ratio	-0.443 (0.371)	-0.194 (0.483)	-0.623 (0.423)	-0.247 (0.582)
Percent Unemployed	2.441 (1.800)	0.154 (0.534)	-3.837** (1.413)	7.074* (2.879)
Median Income	-13.59*** (2.130)	2.404 (1.635)	-11.25*** (1.962)	-24.84*** (4.160)
Percent Immigrant	0.0224 (0.037)	-0.0388 (0.044)	0.0726 (0.044)	-0.0674 (0.046)
Health Professional Shortage	-17.03** (5.571)	0.817 (3.901)	-10.78*** (3.158)	-42.09*** (12.580)
Population Density	0.0211* (0.009)	0.0671* (0.034)	0.0186** (0.007)	0.00158 (0.021)
Gini Index	1.152 (1.058)	-1.184 (0.666)	-0.639 (0.826)	2.623 (1.377)
Constant	245.2** (87.240)	364.6* (150.700)	319.0*** (86.760)	142.1 (119.500)
N	3089	808	1527	754
R <sup>2</sup>	0.676***	0.275***	0.508***	0.562***
AIC	10.81	10.09	9.69	11.25
BIC	8630.89	2799.98	3672.01	3538.50

\* p< .05, \*\* p< .01, \*\*\* p< .001

<b>Table 4.1: Selected Characteristics of Communities by Presence of Reentry Locations</b>			
Variables	Without Reentry Locations	With Reentry Locations	All Counties
Chlamydia Rate (per 100,000)	328.3	544.3***	407.8
Gonorrhea Rate (per 100,000)	71.6	146.0***	99.0
Black Isolation Index Score	19.5	38.9***	26.7
White Isolation Index Score	81.9	69.8***	77.4
% Northeast	17.7	8.7	14.4%
% Midwest	28.5	21.9	26.1%
% South	37.5	35.3	36.7%
% West	16.3	34.1***	22.9%
% College Graduates	21.6	22.2***	21.8
% Latino	10.7	24.2***	15.7
Sex Ratio (Men per 100 Women)	97.3	95.7***	96.7
% Unemployed	7.8	8.5	8.0
Median Income	\$53,573	\$50,154***	\$52,313
% Immigrants	5.9	10.9***	7.7
% with Health Professional Shortage	6.9	0.3***	4.5%
Population Density	218.6	231.2***	223.3
Gini Index Score	43.2	46.5***	44.4
N	2923	166	3089
* p < .05, ** p < .01, *** p < .001			

**Table 4.2:**  
**OLS Regression Models Predicting Chlamydia Rates with Lockup Rate**  
**and Reentry Location, Net of Other Community Factors**

Independent Variables	<b>Model 1</b>	<b>Model 2</b>
Lockup Rate	6.704** (2.344)	-3.796 (5.882)
Reentry Location	218.1*** (26.190)	68.66*** (17.5)
Black Isolation		4.385*** (.526)
White Isolation		-7.645*** (.819)
Northeast		-21.49 (33.8)
Midwest		-35.41 (25.14)
South		-83.25*** (23.73)
College Graduates		-1.202 (.729)
Percent Latino		-3.880*** (.984)
Sex Ratio		-1.697 (2.609)
Percent Unemployed		8.552* (3.607)
Median Income		-39.17*** (6.72)
Percent Immigrant		-3.208 (1.687)
Health Professional Shortage		-27.26* (13.03)
Population Density		0.0492* (.0249)
Gini Index		3.753 (2.645)
Constant	322.7*** (8.816)	1132.5*** (315.8)
N	3089	3089

**Table 4.2 (Cont.)**

R <sup>2</sup>	0.185***	0.656***
AIC	13.6	12.7
BIC	17509.8	14494.6

\* p< .05, \*\* p< .01, \*\*\* p< .001

**Table 4.3:**  
**OLS Regression Models Predicting Chlamydia Rates with Lockup Rate and Reentry Location**  
**by Racial Composition of County, Net of Other Community Factors**

	<b>Model 1</b>	<b>Model 2</b>	<b>Model 3</b>
	< .7% Black	.7-10.3% Black	> 10.3% Black
Independent Variables	White Counties	Integrated Counties	Black Counties
Lockup Rate	-16.45 (12.96)	-15.05*** (3.995)	-.423 (13.9)
Reentry Location	26.98 (76.27)	54.79*** (14.57)	66.67* (28.46)
Black Isolation	-7.189 (5.97)	3.746*** (.605)	2.638* (1.317)
White Isolation	-5.165*** (1.437)	-5.660*** (.857)	-6.529*** (1.356)
Northeast	-10.99 (26.16)	-20.17 (20.25)	6.399 (90.85)
Midwest	-31.73 (21.64)	-20.47 (17.36)	-29.53 (92.37)
South	-84.60** (25.84)	-45.21* (20.54)	-90.39 (73.59)
College Graduates	-.62 (.708)	-.58 (.553)	-.146 (1.354)
Percent Latino		-.129 (.856)	-5.312* (2.413)
Sex Ratio	3.725 (2.315)	3.493* (1.618)	-4.588 (6.192)
Percent Unemployed	13.28*** (3.661)	-5.541* (2.369)	18.58** (7.122)
Median Income	7.661 (9.896)	-37.65*** (4.656)	-63.46*** (13.28)
Percent Immigrant	-.669 (8.092)	-2.713 (1.878)	-2.639 (1.641)
Health Professional Shortage	-.207 (14.29)	-41.04*** (12.05)	-51.03 (29.7)
Population Density	-.173 (.22)	.0498* (.0224)	.003 (.053)
Gini Index	-1.487 (2.153)	-1.851 (1.522)	7.779 (4.046)

**Table 4.3 (Cont.)**

Constant	267.6 (215.8)	732.4*** (206.5)	127.00 (679.4)
N	808	1527	754
R <sup>2</sup>	.426***	.618***	.562***
AIC	12.7	11.8	13.08
BIC	4965.3	6958.4	4947.3

\* p< .05, \*\* p< .01, \*\*\* p< .001

**Table 4.4:**  
**OLS Regression Models Predicting Gonorrhea Rates with Lockup Rate and**  
**Reentry Location, Net of Other Community Factors**

Independent Variables	Model 1	Model 2
Lockup Rate	1.998*	-3.409*
	(.94)	(1.574)
Reentry Location	75.04***	21.19**
	(12.25)	(6.662)
Black Isolation		2.354***
		(.196)
White Isolation		-1.971***
		(.365)
Northeast		-20.26*
		(9.597)
Midwest		16.69*
		(8.042)
South		11.53
		(8.534)
College Graduates		-0.0374
		(.259)
Percent Latino		-1.379***
		(.388)
Sex Ratio		0.289
		(.65)
Percent Unemployed		3.739*
		(1.518)
Median Income		-8.561***
		(2.233)
Percent Immigrant		-2.115**
		(.679)
Health Professional Shortage		-14.10**
		(5.324)
Population Density		.0136
		(.008)
Gini Index		2.002*
		(.89)
Constant	69.94***	109.7
	(3.397)	(93.04)
N	3089	3089



**Table 4.4 (Cont.)**

R <sup>2</sup>	.135***	.711***
AIC	11.87	10.7
BIC	11984.56	8315.3

\* p< .05, \*\* p< .01, \*\*\* p< .001

**Table 4.5:**

**OLS Regression Models Predicting Gonorrhea Rates with Lockup Rate and Reentry Location  
by Racial Composition of County, Net of Other Community Factors**

	<b>Model 1</b>	<b>Model 2</b>	<b>Model 3</b>
	< .7% Black	.7-10.3% Black	> 10.3% Black
Independent Variables	White Counties	Integrated Counties	Black Counties
Lockup Rate	-0.641 (2.916)	-4.669** (1.591)	-5.073 (3.125)
Reentry Location	-14.35 (18.51)	16.09** (5.457)	15.89 (10.84)
Black Isolation	-3.262 (2.325)	1.887*** (.209)	1.813*** (.405)
White Isolation	-0.0816 (.505)	-1.410** (.475)	-1.529** (.518)
Northeast	5.397 (5.144)	-0.52 (6.813)	-31.64 (21.17)
Midwest	11.04* (5.518)	22.11** (7.214)	18.03 (24.25)
South	-2.869 (5.714)	20.60* (9.236)	8.338 (17.04)
College Graduates	-0.169 (.172)	0.106 (.22)	0.313 (.431)
Percent Latino		-0.0869 (.476)	-1.872** (.647)
Sex Ratio	2.591* (1.128)	0.893 (.653)	0.643 (1.317)
Percent Unemployed	1.857 (2.147)	-1.512 (.849)	7.622** (2.864)
Median Income	3.234 (3.609)	-6.978*** (1.495)	-19.38*** (4.224)
Percent Immigrant	2.75 (3.085)	-1.458 (.747)	-2.071** (.66)
Health Professional Shortage	-0.0409 (.0566)	-10.08** (-3.666)	-36.55** (11.85)
Population Density	-0.566 (.478)	0.011 (.007)	.003 (.021)
Gini Index	11.67 (49.01)	0.0769 (.655)	4.239** (1.324)
Constant	-0.0174	92.37	-0.0635

**Table 4.5 (Cont.)**

	(.627)	(90.78)	(164.9)
N	808	1527	754
R <sup>2</sup>	.181***	.547***	.611***
AIC	10.2	9.59	11.14
BIC	2926.0	3531.65	3482.39

\* p< .05, \*\* p< .01, \*\*\* p< .001

## APPENDICES

## Appendix A

Table A.1: Correlation Matrix of Variables Used in the Individual-Level Analysis of the Link Between Sexual Network Theory Factors and STDs

	std	bfem	wfem	lfem	othfem	bmale	wmale	lmale	othmale	educat	income10k	age_r	immigrant	married	cohabiting	firstsex	homosex	condomuse	druguse	analsex	concurrent	riskypartner	core	periphery	bridge
std	1.000																								
bfem	0.114	1.000																							
wfem	-0.028	-0.231	1.000																						
lfem	-0.010	-0.137	-0.236	1.000																					
othfem	-0.003	-0.064	-0.111	-0.066	1.000																				
bmale	0.035	-0.108	-0.187	-0.111	-0.052	1.000																			
wmale	-0.051	-0.202	-0.349	-0.206	-0.097	-0.164	1.000																		
lmale	-0.021	-0.125	-0.216	-0.127	-0.060	-0.101	-0.188	1.000																	
othmale	-0.015	-0.059	-0.102	-0.061	-0.028	-0.048	-0.090	-0.055	1.000																
educat	-0.059	-0.051	0.169	-0.157	0.073	-0.072	0.113	-0.188	0.059	1.000															
income10k	-0.073	-0.160	0.090	-0.119	0.012	-0.059	0.182	-0.054	0.025	0.391	1.000														
age_r	-0.045	-0.024	0.039	-0.010	0.004	-0.029	0.029	-0.043	0.011	0.217	0.160	1.000													
immigrant	-0.038	-0.100	-0.223	0.313	0.146	-0.072	-0.199	0.294	0.125	-0.118	-0.090	0.094	1.000												
married	-0.073	-0.127	0.107	0.030	0.039	-0.085	0.003	-0.015	0.019	0.174	0.298	0.361	0.130	1.000											
cohabiting	0.000	-0.024	-0.003	0.057	-0.011	-0.010	-0.044	0.053	-0.013	-0.099	-0.074	-0.049	0.038	-0.288	1.000										
firstsex	-0.078	-0.070	0.033	0.067	0.078	-0.126	0.030	-0.053	0.064	0.308	0.123	0.195	0.176	0.193	-0.097	1.000									
homosex	0.087	0.042	0.183	-0.028	-0.010	-0.070	-0.090	-0.072	-0.041	0.012	-0.052	-0.034	-0.100	-0.095	0.022	-0.117	1.000								
condomuse	-0.006	0.037	-0.124	-0.041	-0.005	0.118	0.009	0.054	0.038	-0.055	-0.079	-0.221	0.011	-0.254	-0.079	-0.009	-0.038	1.000							
druguse	0.067	-0.032	-0.037	-0.111	-0.043	0.067	0.116	0.014	-0.007	-0.070	-0.046	-0.221	-0.163	-0.237	0.030	-0.200	0.179	0.046	1.000						
analsexany	0.037	-0.073	0.032	-0.068	-0.039	-0.024	0.094	0.035	-0.037	0.040	0.034	0.064	-0.097	-0.017	0.056	-0.173	0.207	-0.129	0.149	1.000					
concurrent	0.115	0.052	-0.049	-0.048	-0.012	0.066	0.016	0.002	-0.016	-0.043	-0.079	-0.134	-0.072	-0.230	-0.053	-0.126	0.111	0.058	0.241	0.129	1.000				
riskypartner	0.097	-0.001	-0.029	-0.021	-0.015	0.058	-0.003	0.025	0.005	-0.067	-0.062	-0.026	-0.018	-0.082	-0.010	-0.078	0.101	0.011	0.132	0.088	0.208	1.000			
core	0.076	-0.046	-0.073	-0.059	-0.022	0.131	0.043	0.055	0.000	-0.056	-0.046	-0.121	-0.046	-0.156	-0.048	-0.129	0.029	0.072	0.180	0.105	0.349	0.193	1.000		
periphery	-0.094	-0.002	0.094	0.098	0.034	-0.162	-0.044	-0.055	0.000	0.086	0.099	0.224	0.095	0.333	0.084	0.188	-0.060	-0.140	-0.270	-0.115	-0.474	-0.170	-0.473	1.000	
bridge	0.001	-0.158	-0.233	0.379	0.044	0.014	-0.220	0.347	0.049	-0.154	-0.097	0.003	0.253	-0.023	0.069	-0.029	-0.010	0.006	-0.011	0.031	0.012	0.020	0.026	-0.037	1.000

## Appendix B

Table B.1: Social Location by Sexual Network Interactions

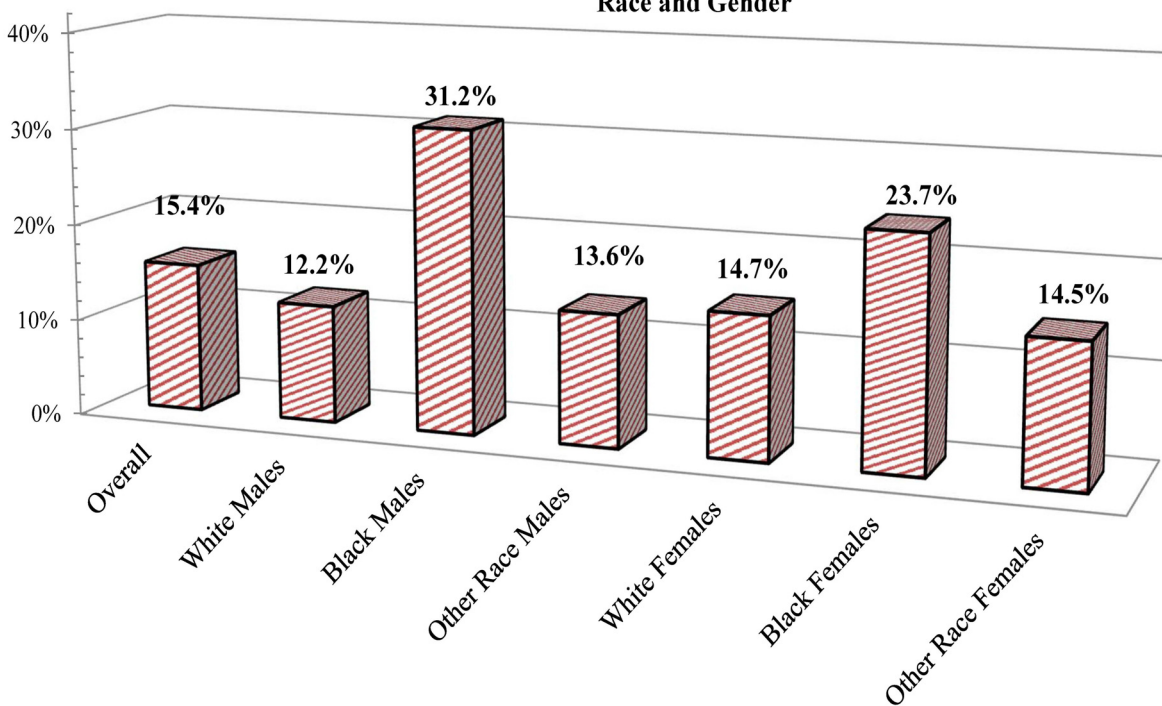
	Social Location							
	Black Women	White Women	Latina Women	Other Race Women	Black Men	White Men	Latino Men	Other Race Men
<b>Sexual Network Characteristic</b>								
<b>Concurrent</b>	Black Women with Concurrent Partners	White Women with Concurrent Partners	Latina Women with Concurrent Partners	Other Race Women with Concurrent Partners	Black Men with Concurrent Partners	White Men with Concurrent Partners	Latino Men with Concurrent Partners	Other Race Men with Concurrent Partners
<b>Not Concurrent</b>	Black Women without Concurrent Partners	White Women without Concurrent Partners	Latina Women without Concurrent Partners	Other Race Women without Concurrent Partners	Black Men without Concurrent Partners	White Men without Concurrent Partners	Latino Men without Concurrent Partners	Other Race Men without Concurrent Partners
<b>Risky Partner</b>	Black Women with Risky Partners	White Women with Risky Partners	Latina Women with Risky Partners	Other Race Women with Risky Partners	Black Men with Risky Partners	White Men with Risky Partners	Latino Men with Risky Partners	Other Race Men with Risky Partners
<b>No Risky Partner</b>	Black Women without Risky Partners	White Women without Risky Partners	Latina Women without Risky Partners	Other Race Women without Risky Partners	Black Men without Risky Partners	White Men without Risky Partners	Latino Men without Risky Partners	Other Race Men without Risky Partners
<b>Core Member</b>	Black Women with Core Membership	White Women with Core Membership	Latina Women with Core Membership	Other Race Women with Core Membership	Black Men with Core Membership	White Men with Core Membership	Latino Men with Core Membership	Other Race Men with Core Membership
<b>Not Core Member</b>	Black Women without Core Membership	White Women without Core Membership	Latina Women without Core Membership	Other Race Women without Core Membership	Black Men without Core Membership	White Men without Core Membership	Latino Men without Core Membership	Other Race Men without Core Membership
<b>Peripheral Member</b>	Black Women with Periphery Membership	White Women with Periphery Membership	Latina Women with Periphery Membership	Other Race Women with Periphery Membership	Black Men with Periphery Membership	White Men with Periphery Membership	Latino Men with Periphery Membership	Other Race Men with Periphery Membership
<b>Not Peripheral Member</b>	Black Women without Periphery Membership	White Women without Periphery Membership	Latina Women without Periphery Membership	Other Race Women without Periphery Membership	Black Men without Periphery Membership	White Men without Periphery Membership	Latino Men without Periphery Membership	Other Race Men without Periphery Membership
<b>Racial Bridge</b>	Black Women with Racial Bridging	White Women with Racial Bridging	Latina Women with Racial Bridging	Other Race Women with Racial Bridging	Black Men with Racial Bridging	White Men with Racial Bridging	Latino Men with Racial Bridging	Other Race Men with Racial Bridging
<b>Not Racial Bridge</b>	Black Women without Racial Bridging	White Women without Racial Bridging	Latina Women without Racial Bridging	Other Race Women without Racial Bridging	Black Men without Racial Bridging	White Men without Racial Bridging	Latino Men without Racial Bridging	Other Race Men without Racial Bridging

### Appendix C: Results for STD2

Differential access to health care and treatment may help account for racial and gender disparities in STDs. Moreover, differential patterns of testing for STDs may help explain why African Americans report having STDs at higher levels. In particular, Witt et al. (2013) document that Black patients were more than twice as likely as others to be tested for STDs during routine medical visits. Such higher rates of testing can operate in a paradoxical way. On the one hand, higher testing rates can lead to the diagnosis and treatment for STDs among those who might otherwise go undiagnosed and untreated and, thus, reduce actual infection rates. On the other hand, such practices, when racialized, higher rates of screening would lead to higher rates being reported for Blacks even as actual STD infection rates are declining, and even as they are declining relative to those rates for other racial groups.

Figure C.1 shows that 23.7% of African American women report that they have been tested for STDs within the past year. This compares with 14.7% of White women, 14.5% of other race women, 12.2% of White men, and 13.6% of other race men. The only group reporting higher rates of STD testing is Black men at 31.2%. As mentioned above, this differential pattern of testing can have implications for reporting having an STD. One way of taking differential rates of testing into account is by examining patterns of STD reporting only among those who have been tested within the past year. To accomplish this, I carried out additional analysis of the NSFG data. This time, however, respondents were included in the analysis only if they said that they had been tested for STDs in the past twelve months. Table C.1 presents a correlation matrix of the variables used in the analysis.

**Figure C.1: Percentage of Respondents Tested for STDs within the Past Year by Race and Gender**



Pearson Chi2 (5) = 423.2, Pr = 0.000

Table C.1: Correlation Matrix of Variables Used in the Individual-Level Analysis of the Link Between Sexual Network Factors and STDs

	std2	bfem	wfem	lfem	othfem	bmale	wmale	lmale	othmale	educat	income10k	age_r	immigrant	married	cohabiting	firstsex	homosex	condomuse	druguse	analsexany	concurrent	riskypartner	core	periphery	bridge
std2	1.000																								
bfem	0.108	1.000																							
wfem	-0.033	-0.263	1.000																						
lfem	-0.031	-0.164	-0.220	1.000																					
othfem	0.002	-0.068	-0.091	-0.057	1.000																				
bmale	0.015	-0.180	-0.242	-0.151	-0.063	1.000																			
wmale	-0.044	-0.205	-0.276	-0.172	-0.071	-0.189	1.000																		
lmale	-0.010	-0.143	-0.192	-0.120	-0.050	-0.132	-0.150	1.000																	
othmale	-0.009	-0.060	-0.080	-0.050	-0.021	-0.055	-0.062	-0.044	1.000																
educat	-0.086	-0.058	0.154	-0.120	0.048	-0.077	0.104	-0.105	0.024	1.000															
income10k	-0.083	-0.139	0.052	-0.067	0.010	-0.058	0.171	0.003	0.017	0.337	1.000														
age_r	-0.078	-0.055	-0.088	-0.047	-0.013	0.064	0.096	0.037	0.049	0.255	0.110	1.000													
immigrant	-0.042	-0.094	-0.157	0.251	0.092	-0.067	-0.129	0.246	0.068	-0.045	-0.041	0.101	1.000												
married	-0.069	-0.092	0.068	0.029	0.017	-0.051	-0.010	0.031	0.038	0.116	0.202	0.269	0.120	1.000											
cohabiting	-0.023	-0.033	0.026	0.071	0.007	-0.014	-0.046	0.003	-0.014	-0.072	-0.028	-0.017	0.047	-0.203	1.000										
firstsex	-0.088	-0.029	0.055	0.068	0.050	-0.118	0.026	-0.049	0.019	0.308	0.110	0.179	0.158	0.118	-0.057	1.000									
homosex	0.105	0.043	0.221	-0.028	0.022	-0.130	-0.088	-0.077	-0.041	0.005	-0.079	-0.024	-0.095	-0.069	0.014	-0.068	1.000								
condomuse	-0.030	0.017	-0.166	-0.070	-0.003	0.170	0.003	0.078	0.046	-0.066	-0.062	-0.120	0.011	-0.178	-0.112	0.000	-0.080	1.000							
druguse	0.098	-0.053	0.030	-0.119	-0.011	0.045	0.097	-0.009	-0.025	-0.049	-0.036	-0.191	-0.148	-0.210	-0.029	-0.152	0.197	0.025	1.000						
analsexany	0.042	-0.108	0.034	-0.060	-0.011	-0.070	0.135	0.070	-0.013	0.046	0.047	0.087	-0.040	0.023	0.021	-0.126	0.225	-0.123	0.152	1.000					
concurrent	0.136	0.035	-0.005	-0.071	0.014	0.027	0.027	-0.017	-0.033	-0.014	-0.040	-0.090	-0.073	-0.187	-0.094	-0.097	0.111	0.019	0.253	0.154	1.000				
riskypartner	0.128	-0.020	-0.013	-0.026	-0.018	0.046	0.012	0.007	0.016	-0.092	-0.064	0.007	-0.028	-0.050	-0.021	-0.072	0.141	-0.014	0.173	0.125	0.211	1.000			
core	0.099	-0.090	-0.059	-0.075	-0.002	0.130	0.072	0.043	-0.014	-0.028	-0.016	-0.102	-0.051	-0.137	-0.081	-0.125	-0.008	0.050	0.201	0.142	0.367	0.186	1.000		
periphery	-0.113	0.045	0.058	0.131	0.012	-0.143	-0.064	-0.047	0.013	0.028	0.042	0.162	0.094	0.290	0.131	0.144	-0.033	-0.124	-0.261	-0.124	-0.455	-0.152	-0.465	1.000	
bridge	0.007	-0.202	-0.204	0.356	0.084	-0.009	-0.170	0.318	0.080	-0.110	-0.051	0.045	0.204	-0.016	0.070	-0.028	-0.016	0.015	-0.029	0.034	-0.026	0.003	0.005	-0.010	1.000



Figure C.2 presents the percentage of respondents with STDs among those who have been screened within the last year by race and gender. It shows that 6.9% of White males who have been screened reported having an STD. This compares with 11.7% of Black males, and 5.4% of other race males. The figure also shows that 5.4% of White females who have been screened report having an STD. In comparison, 12.3% of Black females, and 6.3% of other race females have an STD. Generally, these results suggest that there are significant racial and gender differences in the likelihood of having an STD, as these results produce a  $\chi^2$  value of 39.9 with 5 degrees of freedom and a probability value of  $p < .01$ . It should also be noted that for each racial-by-gender group the prevalence of reporting an STD is substantially higher than when including those who have not been screened within the past year (see Figure 2.1). It is also noteworthy that White women have lower reported rates than do White men. These patterns differ from what occurred when including those who have not been screened within the past year (see Figure 2.1).

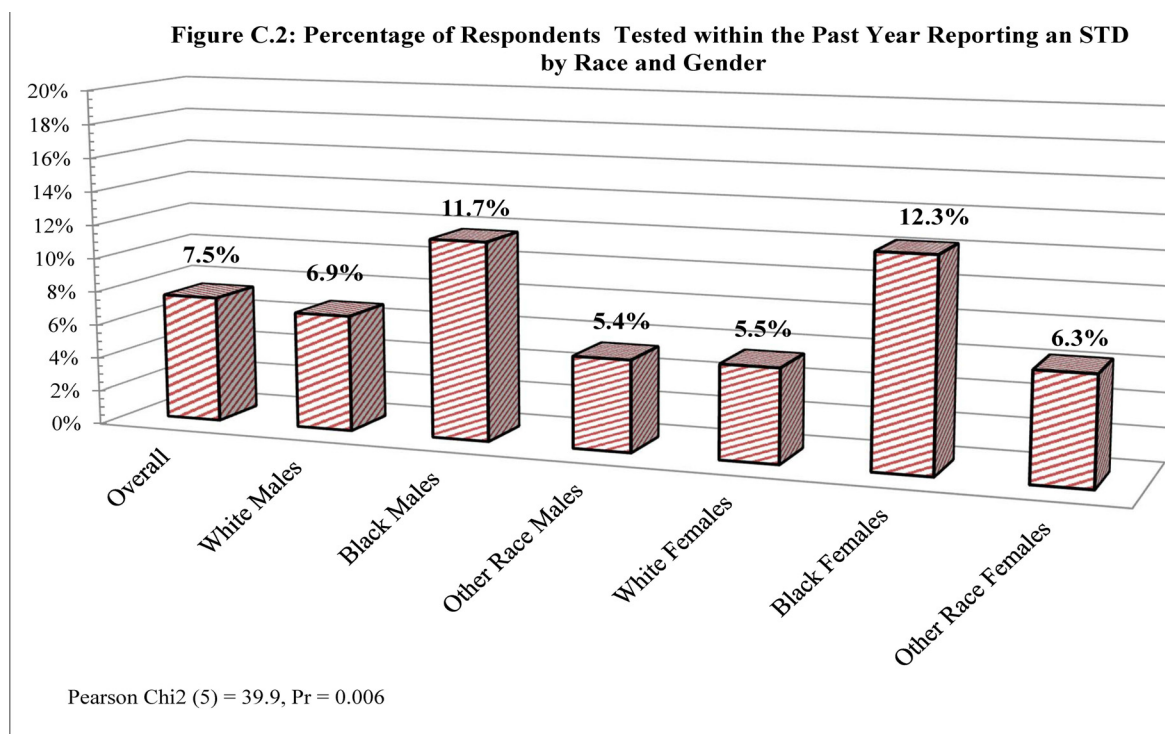


Table C.2 presents the results from logistic regression that predicts the log odds and odds ratios of reporting an STD with social location among those who have been screened for an STD within the past year. Model 1 shows that, among those who have been screened within the past year, the odds of White women reporting an STD are 0.415 times as high as the odds for Black women reporting an STD. Compared with Black women, Latina women have odds that are .302 times as high, other race women have odds that are 1.237 times higher, Black men have odds that are .946 times as high, white men have odds that are .524 times as high, Latino men have odds that are .380 times as high, and other race men have odds of reporting an STD that are .556 times as high. Although the general patterns are similar to those that included respondents who had not been screened within the past year, the relative gap between Black women and other groups is much smaller when looking at only those who have been screened within the past year. Indeed,

there are no statistically significant differences between Black women and Black men, nor between Black women and other race women and men. Model 1 produces a goodness-of-fit test statistic of 0.0 with a p-value of 1.0. This suggests an excellent fit of the data.

**Table C.2**

**Predicting the Likelihood of Reporting an STD among Those Tested Within the Past Year with Social Location, Net of Baseline Factors and Sexual Network Factors**

Independent Variables	Model 1		Model 2		Model 3	
	Coefficient	Odds Ratios	Coefficient	Odds Ratios	Coefficient	Odds Ratios
White Females	-.880***	.415***	-.970***	.379***	-1.017***	.362***
(S.E.)	(.234)	(.097)	(.244)	(.093)	(.248)	(.090)
Latina Females	-1.199*	.302*	-1.272*	.280*	-1.355**	.258**
(S.E.)	(.462)	(.139)	(.527)	(.148)	(.403)	(.104)
Other Race Females	.213	1.237	0.344	1.411	0.222	1.248
(S.E.)	(.570)	(.705)	(.660)	(.932)	(.646)	(.807)
Black Males	-.055	.946	-.016	.984	-.265	.767
(S.E.)	(.251)	(.237)	(.271)	(.267)	(.293)	(.225)
White Males	-.647*	.524*	-.487	.615	-.570*	.566*
(S.E.)	(.269)	(.141)	(.282)	(.173)	(.276)	(.156)
Latino Males	-.968*	.380*	-.958*	.384*	-1.121**	.326**
(S.E.)	(.396)	(.150)	(.438)	(.168)	(.422)	(.138)
Other Race Males	-.587	.556	-.140	.870	-.500	.606
(S.E.)	(.579)	(.322)	(.625)	(.544)	(.594)	(.360)
Education			-.118*	.889*	-.108*	.897*
(S.E.)			(.049)	(.044)	(.051)	(.046)
Income			-.110**	.896**	-.099*	0.906*
(S.E.)			(.040)	(.036)	(.043)	(.039)
Age			-.024	.977	-.021	.979
(S.E.)			(.016)	(.016)	(.017)	(.017)
Immigrant			.070	1.073	.046	1.047
(S.E.)			(.437)	(.469)	(.404)	(.423)
Married			.131	1.140	0.372	1.451
(S.E.)			(.303)	(.345)	(.322)	(.467)
Cohabiting			.060	1.062	.281	1.324
(S.E.)			(.222)	(.235)	(.244)	(.323)
Age at First Sex			-.039	.962	.012	1.012
(S.E.)			(.136)	(.130)	(.140)	(.142)

**Table C.2 (Cont.)**

Same Sex			.626*	1.870*	.537*	1.712*
(S.E.)			(.240)	(.448)	(.245)	(.419)
Condom			-.246	.782	-.204	.815
(S.E.)			(.195)	(.152)	(.194)	(.159)
Drug Use			.438	1.549	.206	1.229
(S.E.)			(.223)	(.346)	(.233)	(.286)
Anal Sex			.063	1.065	-0.152	0.859
(S.E.)			(.215)	(.229)	(.208)	(.179)
Concurrent Partner					.568	1.764
(S.E.)					(.293)	(.517)
Risky Partner					.943*	2.568*
(S.E.)					(.368)	(.944)
Core Member					.352	1.423
(S.E.)					(.275)	(.391)
Periphery Member					-.300	.741
(S.E.)					(.300)	(.222)
Bridge					0.167	1.182
(S.E.)					(.252)	(.297)
Constant	-1.962***		.312		.043	
(S.E.)	(.158)		(.698)		(.816)	
N	3823	3823	3823	3823	3823	3823

Model 2 of Table C.2 shows that when baseline factors such as education, income, age, immigrant status, marital status, age at first sex, same sex intercourse, condom use, drug use, and anal sex intercourse are taken into consideration, the STD reporting gaps between Black women and White women and Latina women become somewhat larger, as the odds of White women reporting an STD are .379 times as high as the odds for Black women reporting an STD, and the odds of Latina women reporting an STD are .280 times as high. For other social locations, the reporting STD gap becomes somewhat smaller and statistically non-significant when baseline factors are added to the model. Model 2 produces a goodness-of-fit test statistic of 0.73 with a p-value of .486. This suggests a very good fit of the data.

However, Model 3 of Table C.2 shows that when the sexual network factors are added to the model, the STD-reporting gaps between Black women and all other social locations become larger. In the case of White women, Latina women, White men, and Latino men, these differences are statistically significant ( $p < .05$ ). More specifically, the odds of White women reporting an STD become .362 times as high as the odds for Black women reporting an STD ( $p < .001$ ), and the odds of Latina women reporting an STD become .258 times as high as the odds for Black women reporting an STD ( $p < .01$ ). The STD-reporting gaps between Black women and White men and Latino men become statistically significant ( $p < .05$ ). The odds of White men reporting an STD become .566 times as high as the odds for Black women reporting an STD ( $p < .05$ ), and the odds of Latino men reporting an STD become .326 times as high as the odds for Black women reporting an STD ( $p < .01$ ). The STD-reporting gap between Black women and Black men and other race women and men remain statistically non-significant. Model 2 produces a goodness-of-fit test statistic of 0.73 with a p-value of .484. This suggests a very good fit of the data.

Model 3 of Table C.2 also shows that, net of other factors, people in relationships with people with concurrent partners are not significantly more likely to report an STD than are those without concurrent partners reporting an STD ( $p = .056$ ). This finding is not consistent with Hypothesis 1. The odds of people with risky partners reporting an STD are 2.568 times higher than are the odds for those without risky partners reporting an STD ( $p < .05$ ). This finding provides support for Hypothesis 2. None of the other sexual network factors are systematically related to reporting an STD, net of the sociodemographic and behavioral factors; thus, these results fail to provide support for Hypotheses 3 and 4. Net of other factors, those who are core members are no more likely than those who are not core members to report an STD, and those

who are periphery members are no less likely than those who are not periphery members to report an STD. However, consistent with Hypothesis 5, net of other factors, those who engage in racial bridging are no more likely to have contracted an STD than are those who do not engage in racial bridging. Model 3 produces a goodness-of-fit test statistic of 0.73 with a p-value of .484. This suggests a very good fit of the data.

Among those who have been screened for STDs in the past year, do sexual network factors work in different ways for different social locations as specified in Hypotheses 1-5? Table C.3 provides some answers. Net of other factors, Black women do not differ systematically from other social locations in how having a concurrent partner is related to reports of having an STD. Model 1 shows that, contrary to the expectations associated with sexual network theory, African American women in relationships with people with concurrent relationships ( $\hat{p}=.16$ ) are not significantly more likely to report an STD compared with African American men ( $\hat{p}=.068$ ) nor Whites women ( $\hat{p}=.042$ ) or men ( $\hat{p}=.066$ ) with concurrent partners. This model shows that when the concurrent partner-social location interaction terms are added to the model, the main effects of being in a relationship with a person with concurrent partners remain statistically non-significant. In addition, it shows that the odds of White women reporting an STD are .339 times as high as the odds for Black women reporting an STD. Compared with Black women, Latino women have odds that are .108 times as high, other race women have odds that are 1.125 times higher, Black men have odds that are .712 times as high, white men have odds that are .681 times as high, Latino men have odds that are .356 times as high, and other race men have odds of reporting an STD that are .826 times as high. In this instance, White women and Latina women are significantly less likely to report having an STD than are Black women, but there are no significant differences between Black women and men of any race. The

concurrent partner-social location interactions do not explain the racial and gender disparities in reporting STDs. Model 2 produces a goodness-of-fit test statistic of 0.15 with a p-value of .857.

This suggests a very good fit of the data.

Table C.3

Predicting the Likelihood of Reporting an STD Among Those Tested Within the Past Year with Social Location,  
Net of Baseline Factors, Sexual Network Factors, and Social Location by Sexual Network Interactions

Independent Variables	Model 1			Model 2			Model 3			Model 4			Model 5		
	Concurrent Partners			Risky Partners			Core Member			Periphery Member			Racial Bridge		
	Coefficient	Odds Ratios		Coefficient	Odds Ratios		Coefficient	Odds Ratios		Coefficient	Odds Ratios		Coefficient	Odds Ratios	
White Females	-1.081***	.339***		-1.102***	.332***		-1.029***	.357***		-1.041**	.353**		-1.095***	.335***	
(S.E.)	(.317)	(.108)		(.261)	(.087)		(.267)	(.095)		(.322)	(.114)		(.268)	(.090)	
Latina Females	-1.590***	.204***		-1.976***	.139***		-1.732***	.177***		-.587	.556		-1.813**	.163**	
(S.E.)	(.411)	(.084)		(.365)	(.051)		(.335)	(.059)		(.452)	(.251)		(.541)	(.088)	
Other Race Females	.117	1.125		.157	1.170		.317	1.373		-1.795	.166		-1.823***	.161***	
(S.E.)	(.948)	(1.066)		(.690)	(.807)		(.669)	(.919)		(1.141)	(.190)		(.474)	(.077)	
Black Males	-.342	.710		-.336	.714		-.257	.774		-.563	.569		-.246	.782	
(S.E.)	(.426)	(.302)		(.326)	(.233)		(.339)	(.263)		(.368)	(.210)		(.339)	(.265)	
White Males	-.385	.681		-.497	.609		-.626	.535		-.506	.603		-.685*	.504*	
(S.E.)	(.352)	(.240)		(.283)	(.172)		(.317)	(.170)		(.404)	(.244)		(.288)	(.145)	
Latino Males	-1.032	.356		-1.145*	.318*		-.912*	.402*		-1.683**	.186**		-.995	.370	
(S.E.)	(.590)	(.210)		(.522)	(.166)		(.457)	(.184)		(.537)	(.100)		(.555)	(.205)	
Other Race Males	-.191	.826		-.543	.581		-.572	.564		-2.134	.118		-2.755***	.064***	
(S.E.)	(.703)	(.581)		(.620)	(.360)		(.668)	(.377)		(1.124)	(.133)		(.629)	(.040)	
Education	-.110*	.896*		-.116*	.891*		-.110*	.896*		-.117*	.889*		-.114*	.892*	
(S.E.)	(.052)	(.047)		(.053)	(.047)		(.052)	(.046)		(.051)	(.045)		(.053)	(.047)	
Income	-.100*	.905*		-.100*	.905*		-.103*	.902*		-.101*	.904*		-.097*	.907*	
(S.E.)	(.043)	(.039)		(.043)	(.039)		(.043)	(.039)		(.043)	(.039)		(.043)	(.039)	
Age	-.023	.978		-.021	.979		-.022	.978		-.021	.979		-.024	.977	
(S.E.)	(.017)	(.017)		(.018)	(.017)		(.017)	(.017)		(.017)	(.017)		(.017)	(.017)	
Immigrant	.073	1.075		.048	1.049		.073	1.076		.013	1.013		.078	1.082	
(S.E.)	(.415)	(.446)		(.421)	(.442)		(.378)	(.406)		(.393)	(.398)		(.410)	(.444)	
Married	.372	1.451		.367	1.443		.372	1.451		.360	1.434		.433	1.541	
(S.E.)	(.315)	(.457)		(.333)	(.481)		(.326)	(.474)		(.318)	(.457)		(.321)	(.495)	
Cohabiting	.310	1.363		.267	1.306		.291	1.338		.301	1.351		.305	1.357	



Table C.3 (Cont.)

(S.E.)	(.247)	(.337)	(.248)	(.324)	(.253)	(.338)	(.255)	(.344)	(.240)	(.325)
Age at First Sex	.012	1.012	.010	1.011	.026	1.027	.036	1.037	.045	1.046
(S.E.)	(.146)	(.148)	(.150)	(.152)	(.140)	(.144)	(.139)	(.144)	(.137)	(.143)
Same Sex	.510*	1.665*	.522*	1.686*	.517*	1.677*	.495	1.641	.535*	1.707*
(S.E.)	(.241)	(.401)	(.233)	(.392)	(.246)	(.412)	(.257)	(.422)	(.246)	(.421)
Condom	-.216	.806	-.245	.783	-.235	.790	-.221	.801	-.211	.810
(S.E.)	(.189)	(.152)	(.202)	(.158)	(.206)	(.163)	(.196)	(.157)	(.190)	(.154)
Drug Use	.197	1.217	.201	1.223	.198	1.219	.184	1.202	.241	1.272
(S.E.)	(.232)	(.283)	(.234)	(.285)	(.233)	(.284)	(.233)	(.280)	(.232)	(.295)
Anal Sex	-.167	.846	-.163	.850	-.151	.860	-.106	.900	-.142	.867
(S.E.)	(.208)	(.176)	(.203)	(.172)	(.204)	(.176)	(.203)	(.183)	(.209)	(.182)
Concurrent Partner	.599	1.821	.568	1.764	.575	1.777	.592*	1.807*	.568	1.764
(S.E.)	(.472)	(.859)	(.297)	(.524)	(.294)	(.522)	(.290)	(.524)	(.292)	(.516)
Risky Partner	.945*	2.572*	.091	1.095	.928**	2.529**	.927*	2.526*	.932*	2.539*
(S.E.)	(.370)	(.951)	(.575)	(.630)	(.347)	(.877)	(.379)	(.959)	(.368)	(.936)
Core Member	.358	1.43	.324	1.383	.086	1.090	.343	1.409	.370	1.447
(S.E.)	(.273)	(.390)	(.274)	(.379)	(.676)	(.737)	(.284)	(.400)	(.271)	(.391)
Periphery Member	-.326	.722	-.318	.727	-.290	.748	-.505	.603	-.311	.733
(S.E.)	(.293)	(.212)	(.301)	(.219)	(.297)	(.222)	(.432)	(.261)	(.303)	(.222)
Bridge	.145	1.156	.148	1.159	.136	1.146	.151	1.163	-.640	.527
(S.E.)	(.257)	(.297)	(.248)	(.287)	(.249)	(.285)	(.250)	(.291)	(.723)	(.381)
White Female X Sex. Network	.154	1.167	1.122	3.071	.237	1.268	.037	1.038	.712	2.037
(S.E.)	(.597)	(.697)	(.743)	(2.282)	(.855)	(1.085)	(.486)	(.504)	(.919)	(1.872)
Latina Female X Sex. Network	.703	2.019	3.563***	35.273**	2.230*	9.299*	-1.247**	.287**	1.248	3.483
(S.E.)	(.574)	(1.158)	(.959)	(33.843)	(.947)	(8.809)	(.441)	(.127)	(.995)	(3.466)
Oth. Race Female X Sex. Network	.233	1.262	2.731**	15.355**	-.607	.545	2.723*	15.228*	3.263**	26.127**
(S.E.)	(1.406)	(1.775)	(.930)	(14.273)	(1.569)	(.855)	(1.296)	(19.739)	(1.037)	(27.097)
Black Male X Sex. Network	.144	1.154	.994	2.702	.212	1.236	.662	1.94	.355	1.427
(S.E.)	(.598)	(.691)	(.672)	(1.816)	(.709)	(.876)	(.598)	(1.159)	(.960)	(1.369)
White Male X Sex. Network	-.508	.602	-.786	.456	.412	1.510	-.273	.761	.979	2.661

Table C.3 (Cont.)

[illegible]

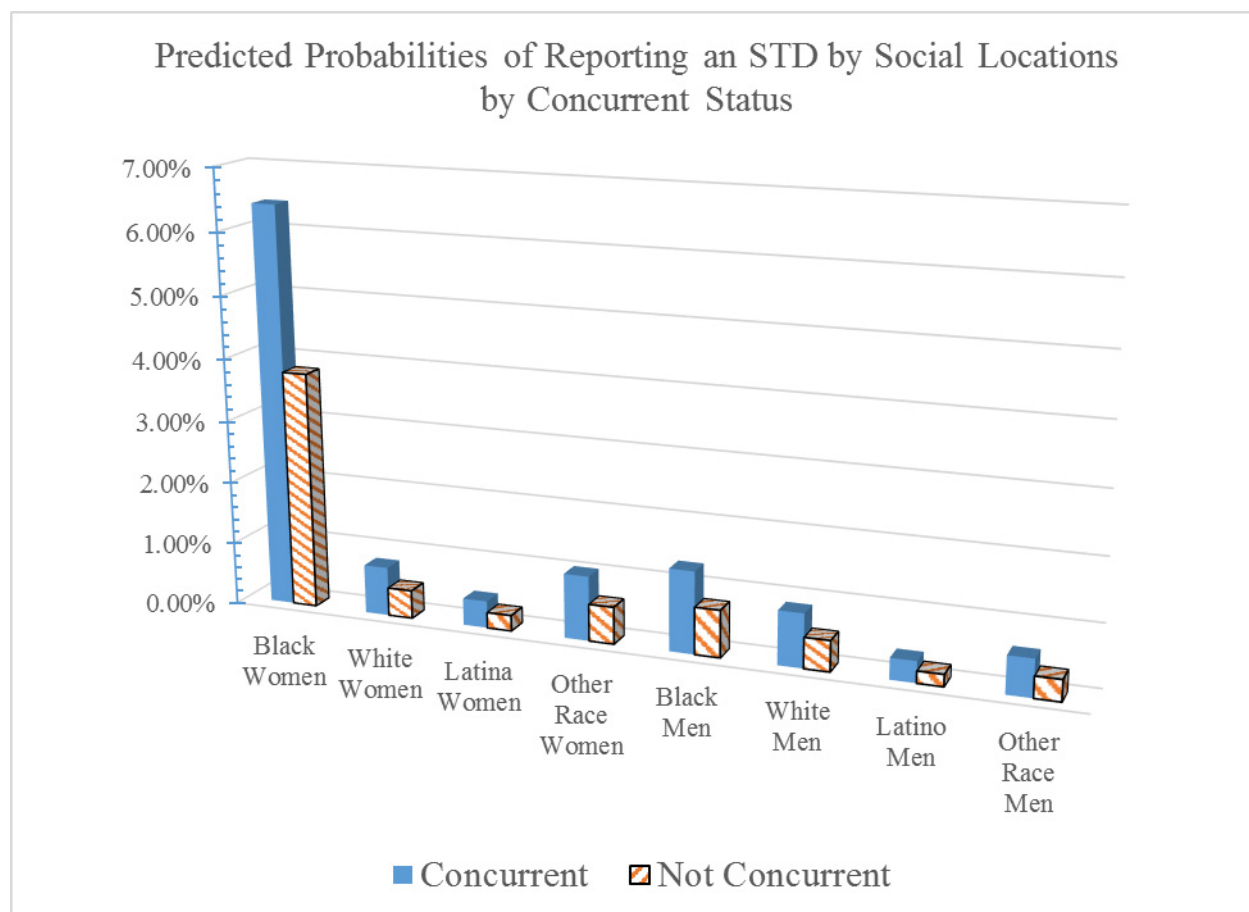
Model 2 of Table C.3 shows that, contrary to the expectations associated with sexual network theory, African American women in relationships with risky partners ( $\hat{p}=.117$ ) are not more likely to report an STD compared with White women ( $\hat{p}=.028$ ) nor men ( $\hat{p}=.066$ ) with concurrent partners. African American women in relationships with risky partners are, however, significantly more likely than are Latina women with risky partners ( $\hat{p}=.0112$ ) to report an STD. This model shows that when the risky partner-social location interaction terms are included, the main effect of being in a relationship with a risky partner becomes statistically non-significant. These results do not support Hypothesis 2, they are contrary to the expectations associated with sexual network theory, and do not explain the racial and gender disparities in reporting STDs.

Do the relationships between these sexual network types and reporting an STD differ by social location? As Model 3 in Table C.3 shows, net of other factors, core members are no more likely to report an STD than are non-core members, and African American core members are no more likely to report an STD than are White core members. These findings do not support Hypothesis 3, and they do not explain the racial and gender gaps in reporting STDs. It should be noted, however, that Latino women with core membership ( $\hat{p}=.0142$ ) are significantly less likely than are Black women with core membership ( $\hat{p}=.114$ ) to report an STD. Similarly, Model 4 shows that, net of other factors, periphery members are no less likely to report an STD than are non-periphery members. Black periphery members ( $\hat{p}=.0356$ ) are no more likely to report an STD than are White periphery members ( $\hat{p}=.0057$ ). Again, however, Latina women with periphery membership ( $\hat{p}=.0288$ ) and other race women with periphery membership ( $\hat{p}=.0085$ ) are significantly less likely to report STDs than are Black women with periphery status ( $\hat{p}=.0298$ ). These findings do not support Hypothesis 4, and they do not explain the racial and gender gaps in reporting STDs.

Does racial bridging operate in the fashion predicted by sexual network theory? Model 5 in Table C.3 shows that racial bridging works differently for other race women than it does for Black women. In particular, other race women involved in racial bridging ( $\hat{p}=.0844$ ) are likely to report STDs than are Black women involved in racial bridging ( $\hat{p}=.116$ ). There are no statistically significant differences in the patterns for Black women relative to White women, Latina women, Black men, White men, Latino men, nor other race men. Thus, they are not consistent with Hypothesis 5. Moreover, these racial bridging-social location interactions do not account for the racial and gender differences in reporting STDs.

## APPENDIX D

Figure D.1



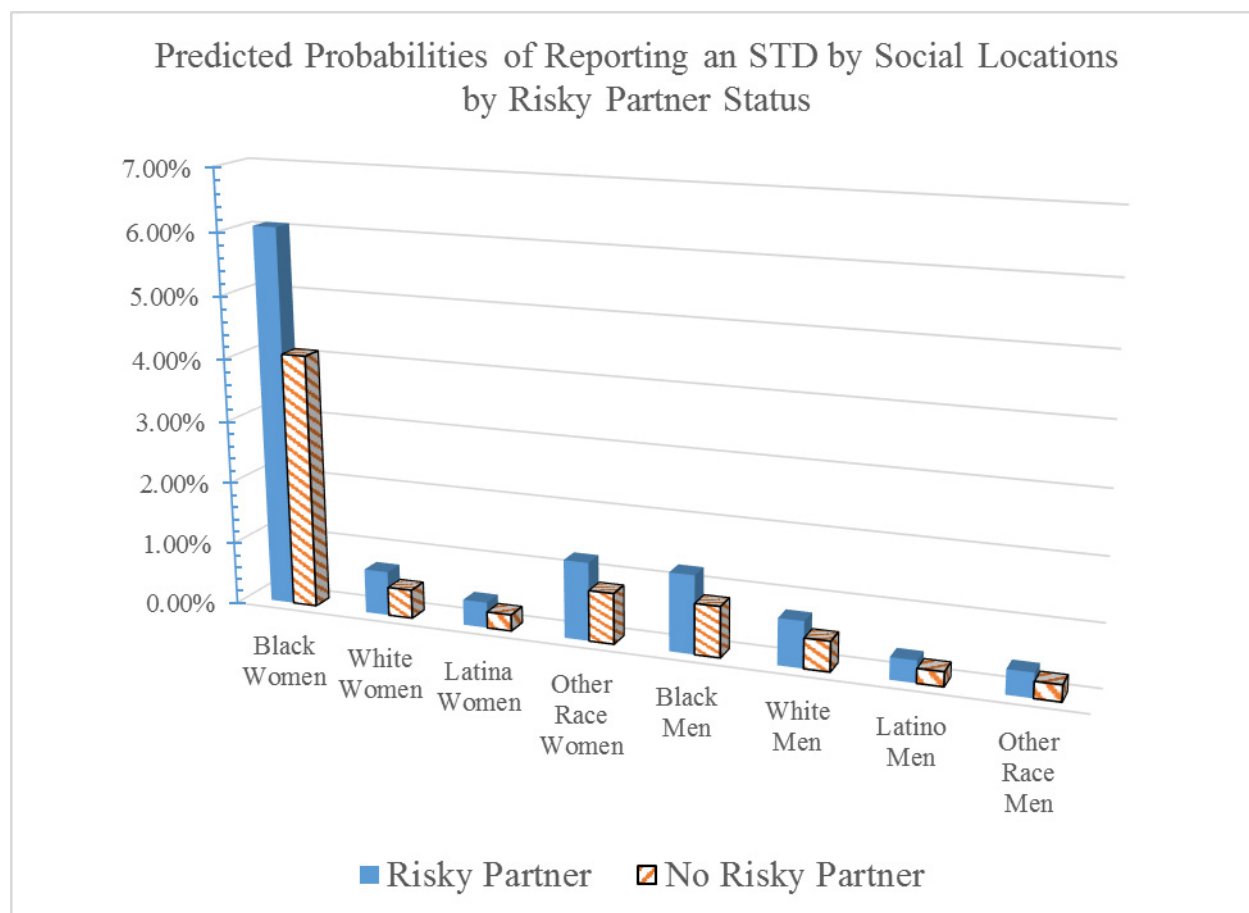
**Figure D.2**

Figure D.3

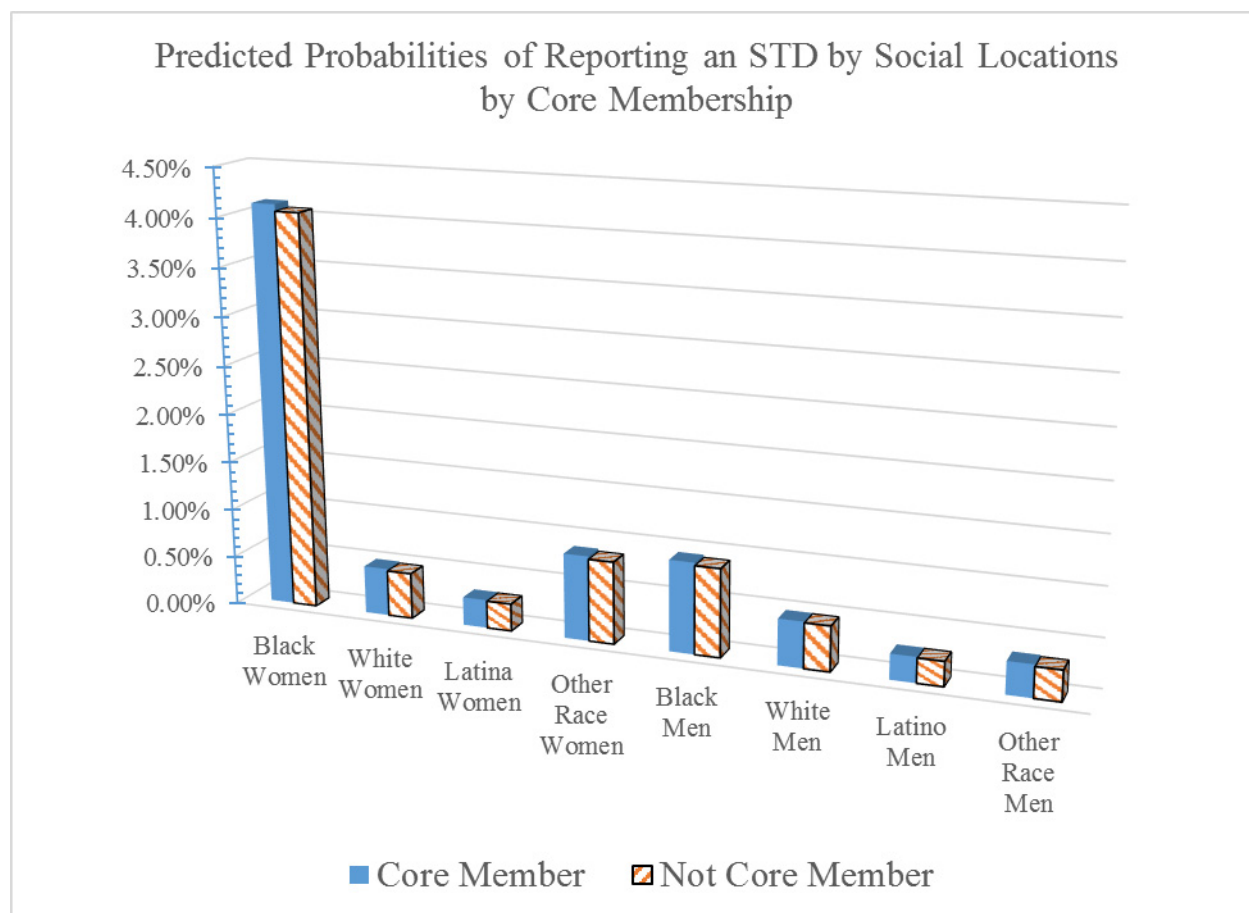


Figure D.4

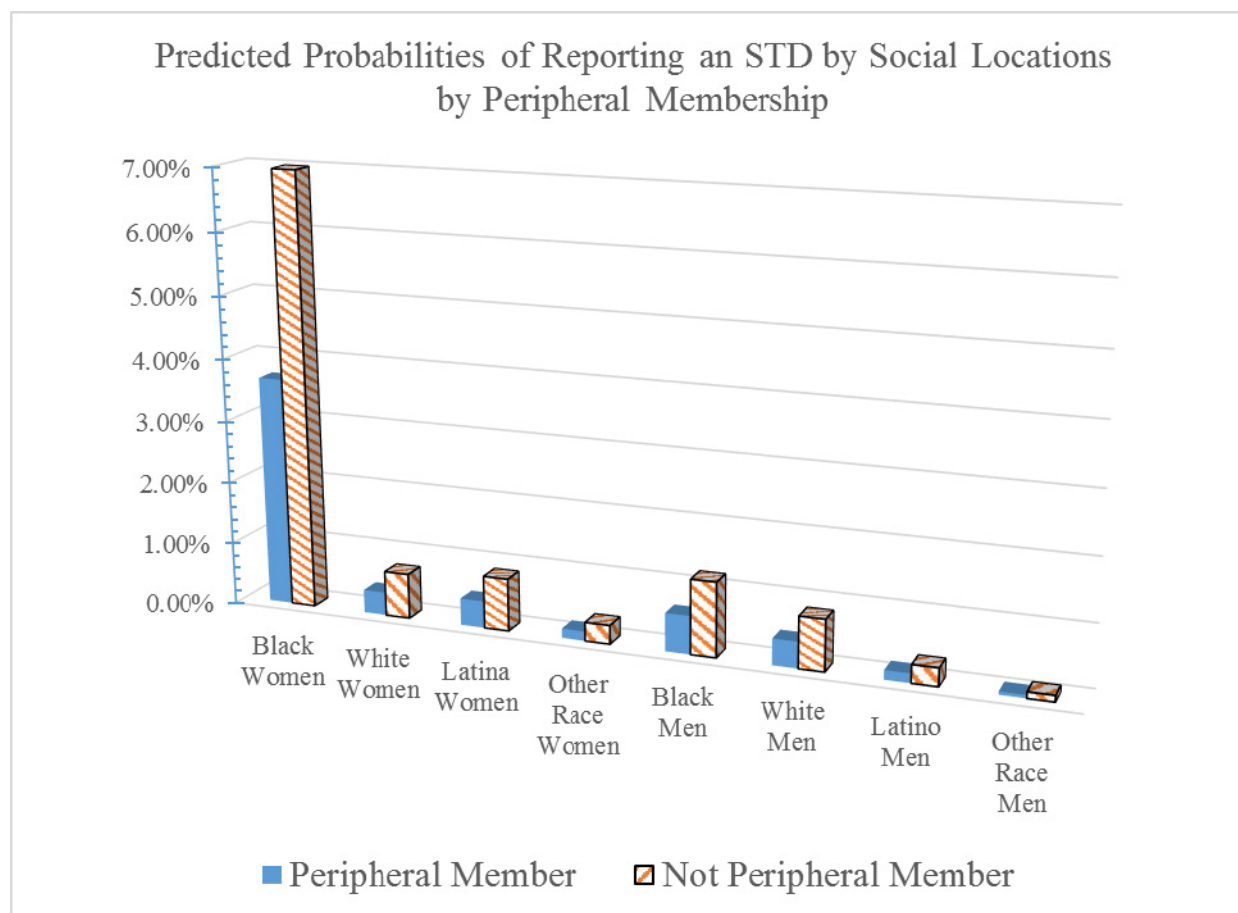
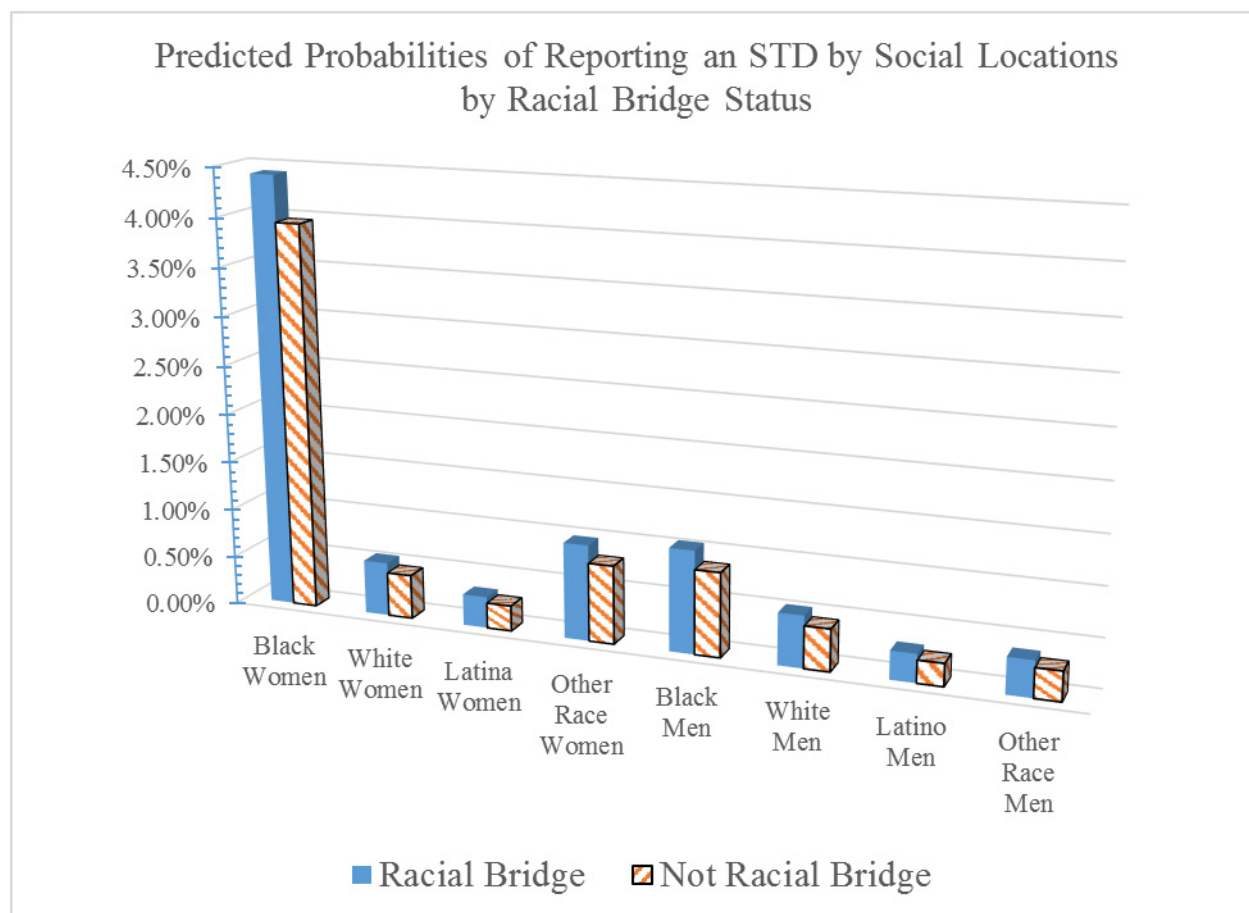




Figure D.5



## Appendix E

Table E.1: Correlation Matrix of Variables Used in Analysis of the Link Between Residential Segregation and STDs

	Chlamydia Rate	Gonorrhea Rate	Black Isolation	whiteiso Isolation	Health Prof. Shortage	% College Grad	% Latino	Sex Ratio	% Unemp.	Median Income	% Immigrant	Population Density	Gini	% High Poverty	County Race
chlamrate	1.000														
gonrate	0.872	1.000													
isolation	0.682	0.770	1.000												
whiteiso	-0.438	-0.277	-0.306	1.000											
healthprof shortage	-0.016	-0.017	-0.065	-0.010	1.000										
collgrad	-0.124	-0.172	-0.071	-0.133	-0.125	1.000									
perlantino	0.160	-0.027	0.019	<b>-0.786</b>	-0.083	0.113	1.000								
sexratio	-0.290	-0.329	-0.421	0.008	0.170	-0.012	0.076	1.000							
perunemployed	0.464	0.419	0.367	-0.268	0.066	-0.127	0.147	-0.099	1.000						
income10k	-0.354	-0.338	-0.145	0.014	-0.190	0.442	0.015	0.004	-0.464	1.000					
perimmigrant	0.098	0.067	0.062	-0.072	-0.008	0.056	0.018	0.045	0.047	0.002	1.000				
popdensity	-0.057	-0.043	-0.063	0.114	-0.157	0.060	-0.055	-0.091	-0.094	0.156	-0.003	1.000			
gini	0.465	0.424	<b>0.508</b>	-0.394	-0.051	0.051	0.320	-0.291	0.232	-0.183	0.053	-0.149	1.000		
highpov	0.068	0.082	0.070	-0.162	0.087	-0.122	0.104	0.015	0.061	-0.164	-0.019	-0.051	0.139	1.000	
countytrace	0.538	0.618	0.763	-0.246	-0.054	-0.032	-0.052	-0.321	0.244	-0.048	0.053	-0.027	0.332	0.052	1.000

## Appendix F

Table F.1: Correlation Matrix of Variables Used in Analysis of the Link Between Incarceration and STDs

	chlamrate	gonrate	lockuprate	reentry	isolation	whiteiso	northeast	midwest	south	west	healthprofshortage	collgrad	perlatino	sexratio	perunemployed	income10k	perimmigrant	popdensity	gini	countyrate
chlamrate	1.000																			
gonrate	0.872	1.000																		
lockuprate	0.004	-0.003	1.000																	
reentry	0.457	0.392	-0.097	1.000																
isolation	0.682	0.770	-0.017	0.411	1.000															
whiteiso	-0.438	-0.277	-0.053	-0.391	-0.306	1.000														
northeast	-0.050	-0.139	-0.063	-0.126	0.085	0.091	1.000													
midwest	-0.040	0.078	-0.034	-0.074	0.022	0.435	-0.247	1.000												
south	0.117	0.254	0.089	-0.017	0.213	-0.180	-0.310	-0.448	1.000											
west	-0.050	-0.253	-0.014	0.202	-0.337	-0.324	-0.227	-0.328	-0.411	1.000										
healthprofshortage	-0.016	-0.017	0.167	-0.156	-0.065	-0.010	-0.070	-0.040	0.130	-0.048	1.000									
collgrad	-0.124	-0.172	-0.106	0.027	-0.071	-0.133	0.282	-0.284	-0.195	0.282	-0.125	1.000								
perlatino	0.160	-0.027	-0.016	0.401	0.019	-0.786	-0.043	-0.343	-0.011	0.407	-0.083	0.113	1.000							
sexratio	-0.290	-0.329	0.676	-0.151	-0.421	0.008	-0.208	-0.031	-0.075	0.291	0.170	-0.012	0.076	1.000						
perunemployed	0.464	0.419	0.106	0.157	0.367	-0.268	-0.099	0.009	0.003	0.070	0.066	-0.127	0.147	-0.099	1.000					
income10k	-0.354	-0.338	-0.191	-0.119	-0.145	0.014	0.266	-0.100	-0.242	0.157	-0.190	0.442	0.015	0.004	-0.464	1.000				
perimmigrant	0.189	0.054	-0.101	0.354	0.236	-0.641	0.069	-0.363	0.024	0.293	-0.140	0.252	0.663	-0.038	0.120	0.198	1.000			
popdensity	-0.057	-0.043	-0.102	0.026	-0.063	0.114	-0.010	0.002	-0.001	0.008	-0.157	0.060	-0.055	-0.091	-0.094	0.156	-0.063	1.000		
gini	0.465	0.424	-0.050	0.436	0.508	-0.394	0.112	-0.215	0.106	0.010	-0.051	0.051	0.320	-0.291	0.232	-0.183	0.395	-0.149	1.000	
countyrate	0.538	0.618	0.034	0.260	0.763	-0.246	0.078	-0.085	0.307	-0.326	-0.054	-0.032	-0.052	-0.321	0.244	-0.048	0.174	-0.027	0.332	1.000

## Appendix G

**Table G.1: Summary of Hypotheses and Whether Supported by Results of Analysis**

Summary of Hypotheses	Supported by Results of Analysis?	
	Yes	No
<b>Individual-Level Analysis (Dependent Variable = STD) in Chapter 2</b>		
<i>Hypothesis 1a:</i> Net of individual, behavioral, and other sexual network factors, those who have concurrent partners are more likely to report an STD.	X	
<i>Hypothesis 1b:</i> Compared with African American women with concurrent partners, African American men are no more likely, but White women, White men, Latino women, Latino men, other race women and other race men with concurrent partners are less likely to report an STD, net of individual, behavioral, and other sexual network factors.		X
<i>Hypothesis 2a:</i> Net of individual, behavioral, and other sexual network factors, those who engage in sex with high-risk partners are more likely to report an STD.	X	
<i>Hypothesis 2b:</i> African American women with high risk partners are no more likely than are African American men , but more likely to report an STD compared with White women, White men, Latino women, Latino men, other race women and other race men with high risk partners, net of individual, behavioral, and other sexual network factors.		X
<i>Hypothesis 3a:</i> Net of individual, behavioral, and other sexual network factors, core members are more likely to report an STD.		X
<i>Hypothesis 3b:</i> African American women core members are no more likely than are African American men, but are more likely to report an STD compared with White women, White men, Latino women, Latino men, other race women, and other race men who are core members net of individual, behavioral, and other sexual network factors.		X
<i>Hypothesis 4a:</i> Net of individual, behavioral, and other sexual network factors, periphery members are likely to report an STD.	X	

*Hypothesis 4b:* African American women periphery members are no more likely than are African American men, but are more likely to report an STD compared with White women, White men, Latino women, Latino men, other race women, and other race men who are periphery members, net of individual, behavioral, and other sexual network factors. X

*Hypothesis 5a:* Net of individual, behavioral, and other sexual network factors, individuals who engage in racial bridging are no more likely to report an STD compared with those who do not engage in racial bridging. X

*Hypothesis 5b:* African American women who engage in racial bridging are no more likely than are African American men, but are less likely to report an STD, compared with White women, White men, Latino women, Latino men, other race women, and other race men who engage in racial bridging, net of individual, behavioral, and other sexual network factors. X

### **County-Level Analysis (Dependent Variable = Chlamydia) in Chapter 3**

*Hypothesis 6:* Net of other factors, black isolation is associated with increases in chlamydia and gonorrhea rates, but white isolation is associated with lower chlamydia and gonorrhea rates. X

*Hypothesis 7:* In white counties, both black isolation and white isolation are associated with decreases in chlamydia and gonorrhea rates. X

*Hypothesis 8:* In integrated and in disproportionately black counties, black isolation is associated with higher rates of chlamydia and gonorrhea, but white isolation is associated with lower rates of chlamydia and gonorrhea. X

### **County-Level Analysis (Dependent Variable = Gonorrhea) in Chapter 3**

*Hypothesis 6:* Net of other factors, black isolation is associated with increases in chlamydia and gonorrhea rates, but white isolation is associated with lower chlamydia and gonorrhea rates. X

*Hypothesis 7:* In white counties, both black isolation and white isolation are associated with decreases in chlamydia and gonorrhea rates. X

*Hypothesis 8:* In integrated and in disproportionately black counties, black isolation is associated with higher rates of chlamydia and gonorrhea, but white isolation is associated with lower rates of chlamydia and gonorrhea. X

**County-Level Analysis (Dependent Variable = Chlamydia) in Chapter 4**

*Hypothesis 9:* As incarceration rates in counties increase, the rates of chlamydia and gonorrhea will increase. X

*Hypothesis 10:* Net of other factors, counties with reentry facilities will have higher rates of chlamydia and gonorrhea compared with counties without reentry facilities. X

**County-Level Analysis (Dependent Variable = Gonorrhea) in Chapter 4**

*Hypothesis 9:* As incarceration rates in counties increase, the rates of chlamydia and gonorrhea will increase. X

*Hypothesis 10:* Net of other factors, counties with reentry facilities will have higher rates of chlamydia and gonorrhea compared with counties without reentry facilities. X